

**DIFFERENTIAL DIAGNOSIS FOR
PRACTITIONERS IN THE TROPICS
A CLINICAL HANDBOOK**

by **W H Jopling**

M R C P (Lond) M R C P (Edin) D T M & H (Eng)

in collaboration with

Sir Philip Manson Bahr

Scientific Department of
FARBFABRIKEN BAYER AG
LEVERKUSEN / GERMANY
Printed May 1959

The main object of this publication is to act as a guide for the general practitioner in the tropics when faced as he usually is with clinical problems in out of the way stations where he is far removed from professional assistance and quite out of reach of the ancillary services which are provided by and considered essential to more populous centres. It is abundantly recognized that clinical diagnosis *per se* is apt to be a much more complicated and tedious task in the tropics than it is in most temperate countries mainly because of the prevalence of many tropical parasitic infections which may be met either in their typical forms or grafted sometimes upon some other disease entities.

We have given this subject considerable attention based upon long personal experience of diagnosis derived from clinical phenomena.

We realize that the methods of examination of the patient may be limited so that the practitioner has to rely upon his own powers of observation assisted by such simple aids as his stethoscope, blood pressure manometer, patellar hammer and clinical thermometer – and his own senses. We realize too that the difficulties of the task before us are considerable because in the tropics microscopic diagnosis is so important especially in connection with parasites of the blood, urine or alimentary canal. We have therefore given attention to the methods by which these difficulties may be to some extent surmounted.

We propose therefore as the most practical method of dealing with this difficult and intricate problem to describe the examination of the patient with emphasis on inspection and palpation.

Philip Manson Bahr

CONTENTS

	7
General Appearance and Manner	8
Gait	10
Voice and Speech	11
Complexion and Facial Expression	15
Scalp and Body Hair	18
Eyelids	21
Eyes	26
Ears	29
Mouth and Pharynx	36
Nose	41
The Neck	47
Chest (Thorax)	50
Examination of the Heart	60
Examination of the Lungs	66
The Sputum	67
Examination of the Breasts	68
The Back	71
Examination of the Abdomen	88
Groins Perineum and External Genitalia	95
Hands	103
The Nails	105
Feet	110
Legs	116
Body Temperature	
Appendix	127
1 Rashes	138
2 Urine	140
3 Faeces	143
4 Laboratory Tests	154
Index	

GENERAL APPEARANCE AND MANNER

A patient who is embarrassed on shaking hands because of delay in relaxing his grasp is suffering from dystrophia myotonica (myotonia atrophica). Look for weakness and wasting of the facial muscles and of the sternomastoids, ptosis, frontal baldness, and slowness in movement due to difficulty in relaxing limb muscle.

While interrogating the patient in the consulting room, note how he reacts to his symptoms and whether he describes them briefly or in great detail. The patient who gives a detailed account of his symptoms, sometimes with the aid of written notes, is probably suffering from a neurosis. Note such abnormalities as dyspnoea, habit spasms (tics), tremor and choreiform movements.

If the patient is in bed, observe how he is lying and whether he is restless or not. A child suffering from headache has a frown on his forehead, and abdominal pain causes him to draw up his knees. A miserable, disconsolate child in the tropics will require investigation for kwashiorkor (malignant malnutrition) – see p. 15. Note the nutritional state of the patient and test for dehydration by examining the tongue for dryness and by pinching up a piece of skin to see if it remains in the abnormal position.

GAIT

Pain producing affections of the legs or feet produce a hobbling or limping gait and examination of the lower limbs will reveal the cause. Common painful conditions in those who have bare feet are injuries and chigger fleas. Those who wear boots or shoes are subject to painful plantar corns or warts. A patient who walks slowly and painfully on the outer border of his feet in a crab like manner is likely to be suffering from secondary yaws and examination of the feet will reveal areas of erosion and fissuring on the skin of the soles. The peculiar gait is adopted in order to avoid the pain which occurs whenever the soles of the feet are subjected to pressure hence the name crab yaws.

When there is a limping gait without obvious cause there is probably some abnormality of one hip. In a child this is likely to be congenital dislocation of the hip, tuberculous disease or a deformity of the head of the femur known as Perthes's disease while an older child or an adolescent may have coxa valga or vara. In an adult who limps it is important to exclude arthritis of the hip. A child with bilateral congenital dislocation of the hip or with bilateral coxa vara walks on a wide base and sways from side to side, the abdomen protrudes and the spine is lordotic. This is known as a waddling gait and lesser degrees of this can be seen in women in the later stages of pregnancy or when suffering from osteomalacia.

Certain diseases of the nervous system have characteristic gaits such as the high stepping which follows foot drop in such diseases as leprosy and beriberi, the ataxic gait in tabes dorsalis, Friedreich's ataxia and in the late stage of African trypanosomiasis, the shuffling short stepped festinant gait in paralysis agitans, the short mincing steps in cerebral arterio sclerosis (*marche à petits pas*), the twisting lurching gait in Huntington's and Sydenham's chorea, the dragging of one foot following a stroke, the scissor's gait in congenital diplegia, the staggering gait in cerebellar disorder and the totally bizarre gait in hysteria. A stiff or spastic gait in the tropics may be due to the lathyrism syndrome (a nutritional disease causing spastic paraplegia) whereas in non tropical countries disseminated sclerosis would be suspected.

A laboured gait occurs in conditions in which the legs are thicker or heavier than normal as for example in elephantiasis and in acromegaly.



VOICE AND SPEECH

The more ill a patient is the fewer words he says and the more softly he speaks. In cholera for instance he can either not speak at all or merely in a whisper.

Impaired articulation may be due to any painful affection of the tongue or jaw but may be an important sign in certain disorders of the nervous system. Slurred speech occurs in acute alcoholism, in dementia paralytica (G.P.I.) in pseudo bulbar palsy and in congenital diplegia. Scanning speech in which every syllable is articulated slowly and deliberately occurs in disseminated sclerosis, Friedreich's ataxia, syringo bulbia and in cerebellar neoplasm.

Oedema of the vocal cords in myxoedema gives the voice a croaking quality. A hoarse voice may have many causes but it is important to exclude inflammatory conditions of the larynx particularly leprosy and tuberculosis or pressure on one recurrent laryngeal nerve as it travels in the neck or the thorax. An otherwise healthy patient who speaks in a whisper and says he is unable to use his normal voice is probably hysterical; this is confirmed by asking him to cough for the cough is phonic in hysteria.

The pitch of the voice varies considerably in different races and individuals. It tends to be higher for example in Chinese than in Africans and the voices of old persons become higher in pitch as the years advance. Vocal pitch is raised in males who have been castrated prior to puberty.

COMPLEXION AND FACIAL EXPRESSION

Pallor occurs in anaemia shock fear anger fainting and associated with vomiting. The doctor should not presume that the patient who is pale is anaemic unless the conjunctivae are pale for some patients with pale complexions have normal blood counts. The complexion becomes muddy in sprue and dusky in kala azar (visceral leishmaniasis).

Jaundice imparts a yellow colouring to the skin and is first manifest in the conjunctivae. The tint of the jaundice tends to be pale lemon yellow in the haemolytic variety orange yellow in toxic (hepatocellular) jaundice and green yellow in obstructive jaundice but these differences may not be obvious in the early stages. In malignant tertian malaria the combination of pallor due to anaemia and yellowness due to mild jaundice gives the skin the appearance of ivory.

Yellowness of the skin may occur in carotenaemia and in patients who have been taking Atebrin[®] (mepacrine). In both these cases the conjunctivae are not involved – compare jaundice – and in carotenaemia the palms are bright yellow. In carotenaemia the palms are more yellow than the backs of the hands but the reverse holds good in Atebrin staining. When Atebrin has been taken regularly over a period of years as a malaria prophylactic the skin of exposed areas such as the face may acquire albeit rarely a blue black colour and dark patches may appear on the oral mucosa particularly on the palate. Also the fingernails become blue. Yellowness of the skin can follow the ingestion of picric acid or its absorption from the broken skin when applied to injuries and burns. It may complicate treatment with santonin and with Miracil D[®] (lucanthone hydrochloride).

Xanthelasma palpebrarum is a benign condition in which yellow patches appear on the inner canthi and eyelids in some middle aged persons. There is no associated hypercholesterol aemia unless diabetes mellitus is present as well – a not uncommon association. Multiple yellow plaques and nodules appear on the skin especially over the joints in xanthoma tuberosum multiplex (cutaneous xanthomatosis) an inherited abnormality in which the serum cholesterol is usually raised. This leads to premature degenerative changes in arteries and cardiac infarction is a common cause of death.



Leprosy
hypopigmented



Pigmentation
types



Vitiligo



Psoriasis

In haemochromatosis haemosiderin in the skin gives a leaden or slate colour. If the adrenals are damaged melanin is deposited in the skin and the combination of haemosiderin and melanin gives the skin the characteristic bronze colour. It is a condition which is much commoner in males than females, has its onset in middle age and is associated with liver enlargement and glycosuria.

The long continued use of silver salts as eyedrops or nasal drops may result in argyria and in this condition the exposed portions of the skin such as the face become a grey blue colour which remains for life.

Cyanosis occurs when there is more than 5 gm of reduced haemoglobin per 100 ml of blood in the capillaries (see p 29). The colour tends to be chocolate blue in methaemoglobinemia and leaden or mauve blue in sulphaemoglobinaemia. Melanin pigmentation (melanosis) may result from external causes such as exposure of the skin to ultraviolet rays, X rays and heat rays or there may be internal causes such as long continued treatment with liquor or enicalis or with oestrogens, cirrhosis of the liver, pellagra, sprue, scurvy, kala azar, chronic malaria, certain reticuloses and lipidoses, a complication of malignant disease known as acanthosis nigricans, scleroderma, urticaria pigmentosa, Addison's disease, von Recklinghausen's disease (multiple neurofibromatosis), renal rickets, subacute bacterial endocarditis and as a complication of normal pregnancy known as chloasma gravidarum.

Leucoderma is a condition in which patches of skin are devoid of pigmentation. Where there has been no antecedent skin disease or disorder the condition is known as vitiligo; the distribution of the depigmented patches is bilateral and often symmetrical and particularly on the arms, hands, legs and feet. Leucoderma may be associated with a number of tropical skin diseases, particularly yaws, pinta, leprosy and post kala azar dermal leishmaniasis. For other signs of late yaws see pages 35, 99 and 111. Post kala azar dermal leishmaniasis (dermal leishmanoid) may occur up to a year or more after completing treatment for kala azar and hypopigmented macules appear on the trunk and limbs, rarely on the face. In addition there may be erythematous or orange coloured patches.

pules and nodules on the face less commonly on other parts of the skin and there may be facial erythema with a butterfly distribution over the nose and cheeks

A peculiar port wine colouration of the face and ears is often but not always present in erythraemia (polycythaemia rubra vera) and there is an associated cyanosis of the tongue and redness of the conjunctivae. Abdominal examination will usually reveal enlargement of the liver and spleen

Spasm of the jaw muscles causing the mouth to be rigidly closed is known as trismus and occurs as an early sign in tetanus. It is followed by spasms of other muscles such as those of the neck and face causing neck rigidity, pursing of the lips or retraction of the angles of the mouth – *risus sardonicus*. Tetanus is not the only cause of trismus for it can be caused by encephalitis and by painful lesions in the region of the jaw. In strychnine poisoning the muscular spasms may at first suggest tetanus but it will be noted that muscular relaxation between paroxysms is complete whereas there is persistent general rigidity between the spasms in tetanus. Tetany is characterised by intermittent muscle spasms in which the hands and feet are commonly involved (see pp 101–102) but other muscles may be affected including those of the face.

SCALP AND BODY HAIR



S p r i n g m d
l p a m t l p s y

In states of malnutrition the hair of the head becomes dull soft and may show changes of pigmentation. The outstanding example of this type of hair change is seen in kwashiorkor (malignant malnutrition) particularly in dark haired children. The short hairs in the temporal region are affected first and become lighter in colour usually from black to brown or even to straw colour. The hair also becomes less plentiful and loses its normal lustre and stiffness. curly hair may become straight.

A patient who is prematurely old with sparse grey hair, a thin and hairless body and loss of libido requires investigation for Simmonds's disease (hypopituitarism). A useful pointer in a male is his story that he had to shave with decreasing frequency. Males who have been castrated before puberty (eunuchs) have a sparse growth of hair on the face and genitals. Pathological greying of the hair may occur in pernicious anaemia and has been known to follow a severe emotional shock. The hair can fall out as a result of typhoid fever but grows again with increased vigour.

The eyebrows become thinned in the lateral portion as a natural process as age advances but madarosis may be an important sign of lepromatous leprosy in the later stages of which the eyebrows may be completely lost together with the eyelashes. Thinning of the lateral aspect of the eyebrow may occur in other conditions chief of which are myxoedema and hypopituitarism.

Bleaching of the hair may result from exposure to sunshine or from the long continued ingestion of high doses of Resochin* (chloroquine)

A condition known as black piedra occurs in parts of South America and the Far East and is characterised by small black nodules developing on the hairs of the scalp. This is caused by a fungus *Piedraia hortai*. Light brown nodules appear on the hairs of the beard or moustache in white piedra, a fungous disease caused by *Trichosporon beigeli*; this occurs in Europe as well as in the tropics. Coloured nodules or concretions occurring in the axillary hairs is diagnostic of trichomycosis axillaris; the nodules are usually yellow, less commonly red or black, and a bacterium known as *Corynebacterium tenuis* is chiefly responsible.

Baldness (alopecia) may be a normal accompaniment of advancing age; in young adults it may be hereditary. Alopecia areata is characterised by round or oval bald patches on the scalp in the absence of any local or general disease or infection. The short hairs at the margins of the patches are thin in circumference where they emerge from the skin and are



normal in circumference further away thus resembling exclamation marks. The cause is not clearly understood but there is usually an emotional disturbance in the background. Rarely the bald patches fuse and eventually the whole scalp becomes bald – alopecia totalis. The eyebrows and eyelashes may also be lost.

Fungal infections causing alopecia are favus and ringworm. In favus the affected areas of the scalp become covered with sulphur yellow cups 2–3 millimetres in diameter each surrounding a hair where it emerges from the scalp. All ages are affected and the scalp has a mouse like odour. Ringworm of the scalp (*tinea tonsurans*) affects children predominantly. In microsporon infections one or more oval or circular bald patches appear the skin being scaly and covered with broken hairs. In trichophyton infections there are no scales and the hairs tend to break off flush with the scalp leaving black dots on the bald patches – black dot ringworm. In chronic lupus erythematosus red scaly patches appear on the scalp later to become smooth hairless scars surrounded by a narrow red scaly margin. The skin of the face may show similar lesions on cheeks and bridge of nose – butterfly distribution and the ears may be scarred and deformed. Lesions of the buccal and nasopharyngeal mucosa should be looked for (see Mouth). In secondary syphilis the scalp may show areas of baldness particularly at the back and sides of the head the hair is not completely lost giving a characteristic moth eaten appearance. Other signs of secondary syphilis should be looked for (see Rashes and Mouth). In advanced lepromatous leprosy bald patches may appear as a rare phenomenon which seems to be least rare in Japanese and European patients. These patches may merge to produce large areas of alopecia or even total alopecia. Other signs of lepromatous leprosy will be present such as thinning of eyebrows thickening and nodulation of ears superficial punctate keratitis and other changes which will be described under appropriate headings. In myxoedema the hair of the scalp may appear thinned dry and brittle but this is not always present. The diagnosis can be confirmed by noting other signs of myxoedema such as slow and lethargic temperament cold and dry skin non pitting oedema of the eyelids and croaking voice.

EYELIDS

In blepharitis the margins of the eyelids are bilaterally inflamed. A unilateral suppuration of one eyelid is termed a sty (hordeolum externum) the suppuration occurring in an eyelash follicle. The commonest cystic swelling on one eyelid is a chalazion often incorrectly termed a Meibomian cyst. A rolling inwards of the lid margin is termed entropion and if it affects the upper lid should suggest the possibility of trachoma. A rolling out of the lid margin is known as ectropion and is commonly seen in facial nerve palsy. This may occur in Bell's palsy and in paralysis of the facial nerve from other causes such as leprosy, trauma, poliomyelitis, polyneuritis and meningitis. The uveoparotid syndrome of sarcoidosis is associated with bilateral facial palsy. All these are examples of lower motor neurone paralysis. Upper motor neurone palsy is due to a lesion above the seventh nerve nucleus, i.e. in the pyramidal tract within the brain and only the movements of the lower part of the face are affected; sometimes involuntary emotional movements of the face remain normal.

Oedema of the eyelids may occur in coryza after bouts of coughing or weeping or with inflammation in the vicinity e.g. conjunctivitis, furunculosis, cellulitis etc. It may occur bilaterally together with oedema of the conjunctivae in malignant exophthalmos (exophthalmic ophthalmoplegia) and the oedema may reach such a degree that it causes complete eversion of the conjunctival sac over the eyelid in the form of a large pouch. In African trypanosomiasis transient localised oedemas occur and may involve one eyelid; the patient should be examined for the typical macular eruption (see Rashes), enlarged glands in the posterior triangle of the neck (Winterbottom's sign) and for hyperalgesia over the tibiae and ulnae (Kerandel's sign). In South American trypanosomiasis (Chagas's disease) the earliest sign of infection is unilateral palpebral oedema, conjunctivitis and swelling of the regional lymph glands (Romaña's sign); an allergic reaction in the region where the reduviid bugs deposited their infected faeces while the patient was asleep. Oedema of the eyelids and face associated with fever, muscular pains and gastro-intestinal symptoms occurs in trichiniasis. The eyelids on one side may be

involved in one of the circumscribed swellings of angioneurotic oedema and the patient will give a history of allergy which will make diagnosis simple. If the patient has lived in tropical Africa remember the possibility that these swellings may be Calabar swellings due to infection with the filarial worm *Loa loa*. Although they can occur on the face Calabar swellings chiefly affect forearms and wrists. They appear rapidly and last for one or two days. Sometimes they are large and diffuse so that the forearm or wrist may appear swollen and tense and pressure on one or more peripheral nerves cause paraesthesiae and even temporary weakness of the fingers. The skin over a Calabar swelling – there is usually only one swelling at a time – may irritate slightly but maintains its normal colour. The patient should be asked if he has ever noticed a worm looking like a thread crossing his conjunctiva for this observation is diagnostic of loiasis. Some patients are conscious of something moving under their skin from time to time.

Oedema of eyelids may occur as part of a generalised oedema in cardiac or renal disease or in wet beriberi. In these patients the oedema of the lids is best seen after recumbency owing to the action of gravity.

When oedema affects face, neck and arms but not the leg or lower half of the trunk there is obstruction to the superior vena cava. The most likely causes are aneurysm or mediastinal neoplasm.

A common manifestation of lepromatous leprosy in the early stages of the disease is puffiness of the eyelids which is often associated with a puffy erythema of the face and with slight oedema of the ankles.

Drooping of eyelids (ptosis) is probably congenital if the patient has no other symptom and has noticed the defect all his life. Ptosis may be one of the signs of third nerve paralysis and the doctor should look for external strabismus, inability to move the eye upwards, downwards or inwards and for a dilated pupil which does not contract to light or on attempting to accommodate. In paralysis of the cervical sympathetic drooping of the lid is associated with a small pupil and enophthalmos (Horner's syndrome). When nerve damage is early or slight it may be difficult to tell the difference between these

two types of nerve lesion unless the doctor watches the affected eyelid when the patient is asked to look upwards. In sympathetic ptosis there is normal elevation of the lid but not in third nerve ptosis. Transient ptosis may accompany the headache due to **brain tumour** or to **ophthalmoplegic migraine**. It may also occur in **myasthenia gravis** and weakness of other muscles especially the masseters will occur at the same time. It is never present on waking and can be rapidly abolished by an injection of prostigmine. Ptosis may be **hysterical** if there is no compensatory elevation of the eyebrows and creasing of the forehead and search should be made for other signs of hysteria such as glove and stocking anaesthesia. It should be noted that patients with myasthenia gravis are sometimes considered hysterical particularly as their ptosis is also not associated with any compensatory over activity of the frontalis muscle but this mistake should not occur if it is appreciated that the ptosis of myasthenia is abolished by sleep and by prostigmine. A **partial ptosis** occurs in **tabes** usually bilateral but unequal in degree on the two sides the pupils react to accommodation but not to light (Argyll Robertson pupils). The pupils are usually small and irregular in outline but they may be large if there is associated optic atrophy. Other signs of tabes may be present in the form of loss of tendon reflexes and position sense in the legs impaired pain sensation and there may be a history of lightning pains and of urinary trouble.

EYES

Prominent Eyes Bilateral proptosis (exophthalmos) is a common manifestation of thyrotoxicosis (see p. 46) but it should be remembered that it may occur in the absence of thyroid overactivity in a condition known as malignant exophthalmos (exophthalmic ophthalmoplegia) due to excessive production of the thyrotropic hormone of the anterior pituitary gland. Rare causes include leontiasis ossium, thrombosis of cavernous sinuses and oxycephaly. Unilateral proptosis may occur in orbital cellulitis, cavernous sinus thrombosis and in neoplasm of the optic nerve or of the eyeball. In Hand-Schüller-Christian disease proptosis may be unilateral or bilateral and it should be noted that proptosis in thyrotoxicosis may commence in one eye before the other is similarly affected.

Red Eyes Red conjunctivae can be found in a number of acute febrile illnesses, particularly in leptospirosis and dengue fever and when associated with a flushed and bloated facies can be found in typhus (particularly epidemic and scrub typhus), plague, yellow fever, sandfly fever and heat stroke. The term 'mask facies' has been coined to describe the appearance of the face in sandfly fever.

When a patient reports redness of the eyes in the absence of any systemic disturbance, the most likely cause is conjunctivitis. This may be unilateral or bilateral and the hyperaemia is most marked at the periphery of the conjunctiva (in contrast to the hyperaemia of iritis which is most marked centrally, i.e. in the region immediately surrounding the cornea). The pupil tends to be large and reacts briskly to light and to accommodation (compare iritis). There may be some discharge which has collected at the inner canthus or on the edges of the lids. Purulent conjunctivitis in the newborn infant is likely to be gonococcal. It is important not to miss a diagnosis of trachoma and the conjunctiva should be examined for enlarged follicles which look like grains of boiled sago. This appearance of the conjunctiva lining the inner aspect of the upper lids differentiates trachoma from follicular conjunctivitis in which the upper tarsal conjunctiva is spared. Partial ptosis may occur in trachoma from oedema of the tissues of the

upper lid and later the upper portion of the cornea in contact with the inflamed conjunctiva of the upper lid becomes vascularised (pannus) and entropion may occur

Iridocyclitis may affect one or both eyes and can easily be mistaken for conjunctivitis if the doctor is not experienced in ophthalmology for the patient's symptoms are the same in both conditions namely pain photophobia and lacrimation. The following points should be observed (1) The redness is most marked in the circumcorneal region, the more distant parts being paler. Also the injection is not as brightly red as in conjunctivitis and tends to have a dusky or even a violet hue due to the fact that the dilatation is in the ciliary vessels which lie deeper than the conjunctival vessels. (2) The pupil tends to be small and to react poorly to light and to accommodation also it may be irregular as a result of previous bouts of iritis causing adhesions between the iris and the lens (posterior synechiae) and between the iris and the cornea (anterior synechiae)

Acute iritis and iridocyclitis may complicate a number of diseases tropical and non tropical. Of the tropical diseases the most important are leprosy onchocerciasis relapsing fever and ulcerative conditions of the large bowel. In leprosy it appears to be precipitated by treatment with sulphones and in onchocerciasis by treatment with diethylcarbamazine

The third condition which comes into the differential diagnosis of the red eye is acute **glaucoma**. This may be unilateral or bilateral and the patient has the same triad of symptoms – pain photophobia and lacrimation. The pain is more severe however and radiates from the eye to other structures in the head. Impaired vision is an important symptom and vomiting may occur to confuse the diagnosis with migraine. It should be remembered that glaucoma occurs in the later years of life and is accompanied by increased intra ocular tension. This gives the eyeball a hard feeling when gentle pressure is applied by the index finger with the patient's eye closed. The pupil is dilated and fixed. It is very important not to miss the diagnosis for the sight of the eye can be quickly saved by correct treatment. Atropine drops are absolutely contra indicated as they cause further increase in the tension within the eyeball. In fact acute glaucoma is sometimes brought on by the injudicious use of atropine drops.

Pinguecula This is a yellow triangular patch on the conjunctiva usually on the nasal side and is found in elderly persons who have been exposed to dry climatic conditions (dust wind etc.) Gaucher's disease should be suspected if pingueculae occur in association with splenomegaly in a young person

Pterygium This is a localised vascular thickening of the conjunctiva which encroaches on the cornea if single it is always on the nasal side Like a pinguecula it results from exposure to adverse weather conditions but may occur in adults of any age and particularly in seafarers

The Cornea Keratitis or inflammation of the cornea is not uncommon in the tropics and there are three diseases in which it is pathognomonic These are lepromatous leprosy syphilis and onchocerciasis Keratitis in **leprosy** usually takes the form of superficial punctate keratitis which begins in the upper part of the cornea as a light milky haze punctuated by tiny white spots resembling grains of chalk of various sizes These spots are miliary lepromata Pannus is likely to be present and takes the form of a network of branching blood vessels surrounding the outer edge of the cornea Leprous keratitis is bilateral is very slow to develop and does not give rise to any symptoms apart from visual impairment if the central part of the cornea becomes involved The face should be examined for other evidence of lepromatous leprosy in the form of thinning of eyebrows thickening of ear lobes thickening of the skin and deepening of the lines and wrinkles – particularly on the forehead – nasal ulceration and discharge Keratitis in **syphilis** is a manifestation of congenital infection usually occurring between the ages of 6 and 12 The patient complains of pain lacrimation and photophobia commencing in one eye and later involving both Examination reveals clouding of the cornea usually spreading in from the periphery in a few days the entire cornea is involved and there is considerable impairment of vision New blood vessels arise at the periphery of the cornea (pannus) and extend towards the centre The condition is known as interstitial keratitis and may be accompanied by iritis or iridocyclitis The child should be examined for other stigmata of congenital syphilis such as Hutchinson's teeth (see Teeth) and eighth nerve deafness (Hutchinson's triad – interstitial keratitis Hutchinson's teeth and 8th nerve deafness) Keratitis in **onchocerciasis** occurs in tropical Africa and in Central America It is very slow in its evolution and is without

symptoms in the early stages unless there is associated conjunctivitis. Later the patient develops photophobia, discomfort and varying degrees of visual impairment. Early signs consist of a few small greyish white spots in the superficial layers of the cornea (it is a bilateral condition) best seen if viewed from the side with the light of a torch being shone from the opposite side. They look like spots of candle grease and can be made out clearly with the aid of a torch and corneal loupe. These nummular opacities increase in number as the disease develops and can interfere with vision. Onchocercal pannus may develop and is often triangular in shape, the base resting on the edge of the cornea (the limbus) and the apex towards the centre. In contrast to trachomal pannus, the onchocercal variety does not involve the upper pole of the cornea; its presence considerably increases the chances of blindness developing.

The Eyes in Nutritional Disease The eyes may give important information regarding the nutritional state of the patient. In vitamin A deficiency the conjunctiva may appear dry and lustreless (xerophthalmia) and may show a light brown pigmentation. Bitot's spots may develop on this type of conjunctiva and take the form of small white or brown patches which are usually present on the outer quadrant of the eye; they look like flecks of dried whitewash which have dropped into the eye and have become stuck to the conjunctiva, but are actually heaped up masses of keratinised conjunctival cells. The most serious ophthalmic complication of vitamin A deficiency is keratomalacia, for it leads to blindness if untreated. In this condition milky patches appear in the substance of the cornea, followed by corneal ulceration and secondary infection. The patient should be examined for other evidence of vitamin A deficiency such as a dry, rough, papular condition of the skin over the outer aspects of the upper arms and forearms, known as phrynoderma or toadskin. A history of night blindness (*hemeralopia*) may provide an important clue and is often noted by the patient before any of the above signs occur.

In riboflavin deficiency (ariboflavinosis) the patient may complain of discomfort in the eyes, photophobia, lacrimation and visual fatigue, and examination reveals redness of the sclera which is most marked around the cornea, i.e. at the corneo-scleral junction. The eyelids may appear red and swollen and the eyelashes may be thinned. The face and scrotum should be examined for evidence of a greasy dermatitis and

there may be fissuring and maceration at mucocutaneous junctions e.g. corners of the mouth (angular stomatitis), nares, palpebral fissures, external auditory meatus, prepuce, vulva and anus. Further signs of riboflavin deficiency are described under Lips and Tongue.

Evidence of vitamin C deficiency (scurvy) may be found in the form of a subconjunctival haemorrhage. This appears as a symptomless red patch under the conjunctiva. The patient should be asked if he has had any bleeding from the mouth, nose, urethra or anus and the skin and joints should be examined for evidence of bleeding. Haemorrhage into the skin gives rise to purpura – red or purple spots and patches which do not fade on pressure and look like bruises – while into the joints it causes swelling, pain and stiffness. The gums should be examined (see Teeth and Gums). If a subconjunctival haemorrhage occurs in a patient who has no other evidence of scurvy, other bleeding diseases should be considered as well as trauma, hypertension and pertussis.

External Ocular Muscles

Strabismus If one of the external ocular muscles is paralysed, the patient complains of diplopia on looking in the direction of the pull of the paralysed muscle. Examination of the eye may or may not reveal strabismus (squint), i.e. a failure of the eyes to remain parallel when the patient is asked to look in a particular direction. The causes can be considered under two headings:

A. Damage to the 3rd, 4th or 6th cranial nerve

- 1 Trauma as in fractured skull or gunshot wound
- 2 Stretching due to proptosis
- 3 Infection as in meningitis, encephalitis, trypanosomiasis, diphtheria and syphilis
- 4 Vascular abnormality such as thrombosis or embolism
- 5 Pressure from a neoplasm
- 6 Toxic effect on the nerve resulting from botulism, snake bite and acute alcohol poisoning. Large doses of Atebrin or chloroquine, used in the treatment of certain skin diseases, are rare causes of ocular muscle paralysis and can be included here.
- 7 Vitamin deficiency as in Wernicke's encephalopathy resulting from acute vitamin B₁ deficiency
- 8 Pressure from disease of bone affecting the orbital fissure as in periostitis and Paget's disease

9 Sclerosis of cranial nerve or its nucleus disseminated sclerosis

B Without cranial nerve damage

- 1 Myasthenia gravis
- 2 Ophthalmoplegic migraine
- 3 Hysteria

Here it should be mentioned that the type of squint most commonly encountered in general practice is not due to paralysis of any of the external ocular muscles and is not accompanied by diplopia. It occurs chiefly in children in whom one eye has become lazy owing to defective vision in that eye and the child uses only the sound one for vision. When the eyes are examined together the squint is present in all positions of the eyeballs. It is known therefore as a concomitant squint. Furthermore if the sound eye is covered the affected eye can be made to move fully in all directions showing that there is *no paralysis*.

Nystagmus This is an associated tremor of the two eyes due to loss of postural fixation and may be physiological or pathological. Physiological nystagmus can be seen in railway or car passengers who are looking out of the window while their vehicle is travelling at speed. It also occurs during thermic and rotatory labyrinthine tests. Pathological nystagmus may be congenital or acquired, and the acquired form can have the following causes:

- 1 Occupational This type of nystagmus occurs in those who work in a poor light and has been recorded in underground miners.
- 2 Partial blindness when it has its onset in infancy e.g. albinism, central scotoma, cataract.
- 3 Defect in cerebellum or brain stem This may be due to disseminated sclerosis, vascular abnormality, neoplasm, syringomyelia or syringobulbia or to certain hereditary disorders such as Friedreich's ataxia and Marie's cerebellar ataxia.
- 4 Vestibular or labyrinthine disturbance This may be due to an auditory nerve tumour, to labyrinthitis, to Meniere's syndrome or to poisoning from streptomycin.
- 5 Weakness of ocular muscles as in polyneuritis, myasthenia gravis and various forms of poisoning such as botulism.

The Pupils

Argyll Robertson pupils have been described as a manifestation of tabes (p 20) but they are also a classical sign of dementia paralytica. The patient should be examined for evidence of mental deterioration, slurred speech, an irregular wavy tremor of the lips, slight tremor of the extended fingers, increased tendon reflexes and up going toes. Before making a clinical diagnosis of dementia paralytica the doctor working in tropical Africa must always ask himself if he has adequately excluded trypanosomiasis of the central nervous system.

A condition which may be confused with tabes dorsalis is Adie's syndrome. It occurs in adult females and is characterised by one pupil being larger than the other and not reacting to light. In addition there is absence of ankle jerks and knee jerks. The patients are perfectly normal in every other respect and the cause of the syndrome is unknown.

Abnormal contraction of the pupil is known as miosis and abnormal dilatation of the pupil is called mydriasis. The 3rd cranial nerve is responsible for constricting the pupil and the cervical sympathetic causes dilatation; therefore miosis is likely to occur when the 3rd nerve is irritated or the sympathetic is destroyed and mydriasis is likely to result from destruction of the 3rd nerve or from irritation of the sympathetic. Conditions which cause irritation in the early stages may cause destruction in the later stages and the causes of damage to the 3rd nerve have already been considered under Strabismus. The cervical sympathetic may be irritated or destroyed by an aortic or carotid aneurysm, enlarged glands in the mediastinum or neck, bronchial carcinoma and syringomyelia. Other causes of unilateral miosis are iritis, foreign body in the cornea and the instillation of eserine or pilocarpine eye drops. Other causes of unilateral mydriasis are blindness from optic atrophy, Adie's syndrome (see above) and the instillation of eye drops of atropine, homatropine or cocaine. Bilateral miosis occurs in opium or morphine poisoning, in pontine haemorrhage and as has already been mentioned in Argyll Robertson pupils unless there is associated optic atrophy. Bilateral mydriasis occurs in poisoning from atropine, hyoscine, cocaine or alcohol, in certain mental states such as mania and when bilateral optic atrophy is present.

Grouped vesicles appear around the lips in herpes simplex (herpes febrilis) This is a condition which tends to recur in certain individuals whenever they develop fever from whatever cause in the tropics it is common for herpes simplex to occur in malaria and in relapsing fever

Lesions of chronic lupus erythematosus may appear on the lips where they look like patches of dried collodion

Tongue It is normal to have a certain amount of fur on the tongue but this is increased in toxic conditions in dehydration and in heavy smokers A brown or black fur on the tongue of a healthy person is due to a harmless fungus (*Cryptococcus linguae pilosae*) it sometimes develops in patients who are being treated by antibiotics and is due to the destruction of the bacteria of the mouth which normally keep the growth of oral fungi under control

The tongue assumes a purple or magenta colouring in riboflavin deficiency and may appear similarly coloured in some cases of polycythaemia rubra vera

When the filiform papillae are lost the tongue appears smooth red and devoid of fur This raw beef appearance occurs in certain anaemias in pellagra and in sprue and the patient complains of soreness whenever he drinks anything hot or eats food which is salty or spiced In these patients with smooth tongues the fungiform papillae may appear pink swollen and especially prominent

The appearance of the tongue may be characteristic in scarlet fever On the first day swollen red papillae project through the white fur on the dorsum of the tongue and give the appearance which is known as the white strawberry tongue By the fourth day the fur has peeled off leaving the tongue clean and red with prominent papillae the so called red strawberry or raspberry tongue

Note should be made of the size and muscle tone of the tongue of any inequality in the two sides and of any involuntary movements A small dry tongue is found in dehydration A small flabby tongue showing marked fasciculation (so that it resembles a bag of worms) is diagnostic of progressive

bulbar palsy In pseudobulbar palsy on the other hand the tongue is of normal size but spastic (possessing increased muscle tone) fasciculation is absent

Damage to the twelfth cranial nerve causes hemiatrophy of the tongue on the side of the lesion and when the tongue is protruded it deviates to the atrophied side Another condition in which one half of the tongue becomes wasted is progressive facial hemiatrophy a condition in which wasting of one half of the tongue is associated with atrophy of the same side of the face When protruded the tongue comes out straight Syringobulbia may cause wasting of the tongue unilateral at first

A large tongue is seen in cretinism and in acromegaly

The tongue should be inspected for evidence of ulceration Superficial ulcers with a whitish base are not uncommon and are known as aphthous ulcers They are usually found on the under surface of the tongue and on the buccal mucosa their aetiology is unknown and they often occur in those who are suffering from tropical sprue A carcinomatous ulcer begins as a hard nodule which enlarges and breaks down to form an irregular deep sloughing ulcer with raised and nodular everted edges It is usually situated on the edge of the tongue is painful feels hard on palpation has a surrounding area of induration and limits the mobility of the organ The neck should be examined for the presence of hard enlarged lymph glands Syphilitic ulceration may be primary secondary or tertiary A primary ulcer is usually on the tip and begins as a papule which becomes a small indurated ulcer the submaxillary lymph glands are enlarged In secondary syphilis there are multiple mucous patches and superficial ulcers which may be on the oral and pharyngeal mucosa as well as on the tongue Mucous patches are round or oval patches which are slightly raised and are covered with whitish sodden epithelium ulcers are elongated and snail track in appearance In addition the tongue may have smooth red patches devoid of epithelium Other signs of secondary syphilis should be searched for such as a rash which is macular or papular and is neither irritating nor scaly condylomata in moist regions such as axillae perineum and on the angles of the mouth syphilitic alopecia (see Scalp) and syphilitic onychia (see Nail) Tertiary syphilitic involvement of the

tongue may take one of two forms. The first is known as chronic superficial glossitis in which superficial and irregular ulcers are associated with whitish opaque areas of thickened epithelium (leukoplakia). The second is gummatous ulcer. This begins as a firm swelling in the muscle of the tongue usually in the midline and in the posterior half which later breaks down to form a deep ulcer with irregular punched out walls and wash leather slough at its base. It differs from a carcinomatous ulcer in that it is not painful, it has not the same hardness, there is less surrounding induration, protrusion of the tongue is not limited and there is usually no enlargement of cervical glands. Tuberculous ulceration is usually associated with pulmonary tuberculosis and one or more painful ulcers are situated on the tip or sides of the tongue in its anterior half. The ulcers are small in size, irregular in outline, have undermined edges and are not hard on palpation. A dental ulcer is situated near the sharp edge of a decayed tooth and is produced by trauma from that tooth; it heals as soon as the tooth is removed. An ulcer may develop on the fraenum of the tongue in children suffering from whooping cough. Congenital fissuring of the tongue is a benign condition in which the fissuring is bilateral and symmetrical and the papillae are normal. The geographic tongue is another benign condition with patchy red areas where the epithelium has become denuded in an otherwise normal tongue.

A scarred tongue is suggestive of epilepsy, the scarring having resulted from tongue biting during epileptic convulsions. Tremor of the tongue and lips may occur in nervousness, anxiety, neurosis, thyrotoxicosis, delirium tremens, chronic mercury poisoning, dementia paralytica and in cerebral trypanosomiasis. A protruded tongue which undergoes involuntary bizarre movements is diagnostic of chorea.

The presence of blood blisters on the tongue and oral mucosa of an African is strongly suggestive of onyala – a bleeding disease associated with thrombocytopenia.

The veins on the under surface of the tongue are swollen in congestive heart failure.

Mouth White patches on the mucosa of the mouth, looking like milk curds but not easily removed, occur in a condition



Am. c. l. hman
fth. f. g.

known as thrush and caused by a fungus *Candida albicans*. It is a not uncommon complication of treatment with penicillin or other antibiotics. In lichen planus, a disease of unknown aetiology, white patches appear on the inner aspects of the cheeks opposite the teeth and sometime on the tongue, palate or gums as well. Instead of white patches there may be white striae or discrete pin head papules. Similar lesions may be present on the glans penis or on the labia while the typical flat topped papules of lichen planus occur on the skin. Vesicular pustules in the mouth are found in smallpox.

Patches of brown or black pigmentation on the mucosa of cheeks and gums are commonly found in those with pigmented skins particularly in Africans but when occurring in those with light skins are strongly suggestive of Addison's disease. In such a case it would be important to examine the lungs carefully to exclude tuberculosis. Should an African develop Addison's disease the presence of pigmentation on the tongue would be suggestive as also would the finding of melanoplakia in one whose mouth was known to have been clear of melanotic patches previously. In certain other diseases apart from Addison's disease in which melanosis of the skin becomes

well marked melanoplakia may occur as for example in haemochromatosis. There is an interesting group of patients in whom buccal pigmentation is associated with intestinal polyposis. Intussusception is likely to bring the patient to the doctor.

Ulceration of the buccal mucosa occurs in a number of conditions. Aphthous ulceration has been described under Tongue. In dermatitis herpetiformis (Duhring's disease) there may be small shallow ulcers in the buccal mucosa which are not particularly painful and the skin always provides confirmatory evidence in the form of the typical itching vesicular eruption. In pemphigus vulgaris, a disease affecting middle aged and elderly persons, shallow ulcerations commonly occur in the mouth and they may extend to produce large red areas of denuded epithelium which are very painful and tender. Examination of the skin will reveal the typical bullous eruption of pemphigus vulgaris. Behcet's syndrome, a disease which principally affects young adult males, is characterised by recurrent bouts of ulceration in the mouth and on the genitalia together with iritis. In agranulocytic angina the patient complains of fever, malaise and sore throat and examination reveals ulcerating membranous lesions of the tonsils, palate and gums. The patient should be questioned regarding any recent treatment with drugs liable to cause agranulocytosis such as gold, sulphonamides, amidopyrine, thiouracil and tridione, or his work may have brought him in contact with benzol. Acute leukaemia causes a similar picture. In cancrum oris (noma) the patient is usually a child who is undernourished and suffering from some chronic disease. It is a slowly progressive gangrene of the buccal mucosa which leads to destruction of the tissues of the cheek and is more likely to occur in untreated kala azar than in any other chronic infection owing to the virtual absence of granulocytes in the blood. In South American leishmaniasis (espundia) and in East African leishmaniasis nodules appear on the mucosa of the mouth or nose (or both) which ulcerate and lead to extensive tissue destruction. Parts of the nose and cheek become destroyed causing great disfigurement. Lupus vulgaris may affect the mucosa of nose, mouth and palate either primarily or by spread of infection from the adjacent skin. A primary lesion on the mucosa is a slightly raised red granular patch which becomes ulcerated. If the disease is not arrested there is gradual destruction of tissues such as the nose. In lepromatous leprosy

there may be ulceration of the mucosa of nose, palate and tongue. The nasal cartilage may become destroyed leading to saddle nose and destruction of the palate may cause perforation. In tertiary yaws a condition known as gangosa may occur in which there is slow destruction of the hard palate together with the bones, cartilages and soft tissues of the nose. Other bone lesions of yaws may assist in confirming the diagnosis e.g. sabre tibia, deformed fingers and symmetrical swellings on either side of the nose due to hyperostosis of the nasal processes of the superior maxillae (goundou). In addition the palms and soles should be examined for hyperkeratosis. In bejel, njovera and secondary syphilis mucous patches or plaques appear on the soft palate, tonsils and pillars of the fauces. They are slightly raised patches with a red margin and covered with a milky white layer of superficial necrosis resembling a snail track. Other signs should be looked for such as condylomata around the anus and female genitalia and moist papules at the angles of the mouth. In congenital syphilis radiating scar or rhagades develop at the angle of the mouth.

The mucosa of the mouth is a slate blue colour in argyria (see p. 13) and there are blue black patches of dark pigmentation on the palate in chronic mepacrine poisoning - see p. 11. A greenish discolouration occurs in jaundice.

Teeth and Gums

The teeth have a pink colouration in congenital porphyria and have a dark mottling in fluorosis. Hutchinson's teeth in a child suggest the possibility of congenital syphilis, the affected teeth being the central incisors of the second dentition. These four teeth, especially the upper ones, are broader at the gum margin than at the periphery, giving them a peg shape, and usually there is a notch in the middle of the biting edge. In addition the cusps of the 6 year molars are badly formed and look as if they had been squeezed together to produce a lobulated mulberry appearance.

A blue black line at the gum margin can be caused by the ingestion of lead or mercury and by injections of bismuth.

In gingivitis the gums become red swollen and tender, and a common cause in the tropics is Vincent's infection due to spirochaetes and fusiform bacilli. In pyorrhoea the gums become retracted and pus can be expressed by gently squeezing them against the adjacent teeth. Swollen gums which bleed easily may be a sign of scurvy but it should be noted that gum changes do not occur in those who are edentulous. Further evidence of scurvy should be sought (see p. 25).

Tonsils

The tonsils should always be examined when investigating the cause of fever especially in children the commonest disorder being acute follicular tonsillitis. The tonsils appear red and swollen pus is present in the follicles the tonsillar lymph glands are tender and the patient often has a flushed face with circum oral pallor.

A peritonsillar abscess (quinsy) causes a swelling just above the tonsil which displaces it towards the midline. The practitioner in the tropics should consider the possibility of bubonic plague in a patient suffering from a tonsillar abscess.

Diphtheria should be suspected in a patient who is toxic and ill if a creamy adherent membrane is present over part of one tonsil or more extensively in the fauces. There is a characteristic foetor of the breath and the tonsillar glands in the neck are enlarged but are not particularly tender. Ulceration is absent.

The presence of a tonsillar membrane causing possible confusion with diphtheria may occur in Vincent's angina (fusospirochaetosis) glandular fever (infectious mononucleosis) and in acute leukaemia. In Vincent's angina the breath has a distinctive odour which is predominantly sweet ulceration is present on one or both tonsils the regional glands are only slightly enlarged and tender and the patient does not appear ill or toxic. Tonsillar ulceration may not be apparent until the moderately adherent membrane has been removed. In glandular fever of the anginose type the pseudo membrane is not adherent there is considerable enlargement of the cervical

lymph glands and the patient is less toxic than in diphtheria other groups of lymph glands may become enlarged and the spleen may become palpable. Acute leukaemia should be suspected in an ill and anaemic patient in whom the tonsillar ulceration is part of a more generalized ulcerative condition of the pharynx and gums. the gums appear swollen and haemorrhagic and the tonsillar pseudo membrane if present is not adherent.

Syphilitic changes in the tonsils usually take the form of snail track ulceration as described under Mouth (see p 35) but one tonsil may rarely be the seat of a primary chancre or of a gumma.

Malignant disease of the tonsil should be suspected if there is a deep sloughing ulcer with indurated edges and if the ulcer feels hard on palpation.



NOSE

A characteristic redness and thickening of the skin of the nose and central portion of the face occurs in rosacea

In rhinophyma the nose is swollen and covered with irregular warty vascular nodules and the patient may give a history of long continued over indulgence in alcohol

In rhinoscleroma a condition which has histological and bacteriological similarities to ulcerating granuloma of the pudenda (see p 90) a slowly developing granulomatous process takes place in the nose enlarging the organ and obstructing the airway through it (Hebra nose) The firm pale granulomatous masses may spread into the nasopharynx and even into the trachea

Mention has already been made of nasal destruction in yaws leishmaniasis and in lupus vulgaris also of saddle nose deformity in leprosy (see p 35) A similar deformity can occur in congenital syphilis as a result of destruction of nasal cartilage

The nose is enlarged in acromegaly (see p 98)

A polypoidal affection of the mucous membrane of the nose caused by a fungus is known as rhinosporidiosis the polyps are soft raspberry like and bleed easily



Am 1 hm f th



Am 1 hm d m th



Am 1 hm



Am 1 hm (D C J H d H G (P)

Leishmanial granulomata may develop inside the nasal cavities in oro nasal leishmaniasis causing a bulbous appearance of the nose. Healing with subsequent scarring may give rise to a peculiar deformity of the tip of the nose which is perfectly described in the term *tapir nose* – see photograph on p 39. *This type of leishmaniasis occurs commonly among forest workers in Brazil, Paraguay and Peru (South American leishmaniasis) but may occur less commonly in other parts of the world where cutaneous leishmaniasis is endemic particularly in the Sudan and in Kenya.*

A red scaly rash stretching across the bridge of the nose and on to the cheeks in a butterfly distribution is typical of chronic lupus erythematosus and sometimes there is involvement of the mucosa of the mouth and lips where the lesions resemble patches of dried collodion. Facial erythema with a similar butterfly distribution has already been described under post kala azar dermal leishmaniasis (p 13-14) and pellegra (p 42).

The nares may be the seat of a diphtheric infection the symptom being blood stained nasal discharge. The patient has little in the way of constitutional symptoms but it is important to make a correct diagnosis as he is a dangerous source of infection.

Chronic nasal discharge sometimes blood stained with crusting inside the nares and obstruction of airway frequently occurs in lepromatous leprosy.

One of the earliest symptoms in an infant suffering from congenital syphilis is a mucopurulent nasal discharge (snuffles).

THE NECK

Shortening or spasm of the sternocleidomastoid on one side of the neck gives rise to torticollis a condition in which the head is tilted towards the shoulder on the affected side and rotated towards the opposite side. When occurring in infancy the cause is likely to be a birth injury to the affected muscle but in older children and in adults the most common cause is transient stiff neck (myalgia) a condition which clears up spontaneously in a few days. Should such improvement not occur then care must be taken to exclude hysteria, underlying sepsis or an abnormality of the cervical vertebrae. A sudden and involuntary jerking of the head to one side is likely to be a manifestation of habit spasm (tic) and other muscles are often involved in this repetitive spasmodic movement particularly trapezius and the facial muscle.

Stiffness of the neck is likely to be due to myalgia if it comes on suddenly in an otherwise healthy person. If it lasts more than a few days or has a tendency to recur it is probable that there is an underlying abnormality in the cervical vertebrae and it is necessary to exclude ankylosing spondylitis in a young adult and arthritis (cervical spondylosis) in an older patient. Stiffness of the neck in someone who is febrile and ill calls for exclusion of tetanus and meningitis.

Congenital webbed neck (pterygium colli) may occur alone or may be combined with other anomalies. It is a characteristic feature of Turner's syndrome whether in males (testicular agenesis) or in females (ovarian agenesis) and is associated with a short stocky body, a shield like chest, impotence in males and amenorrhoea in females.

A patient with a short neck, limited head movements and hair growing low down on the neck is suffering from the Klippel Feil syndrome. This is a congenital defect in which the cervical vertebrae are fused.

Hypertrophy of the neck muscles occurs in conditions where respiration is impeded especially in chronic asthma.

Scars on the back of the neck indicative of boils (furunculosis) will require an examination of the urine to exclude diabetes.

The back of the neck is a common site for the dermatitis of pellagra and other skin areas exposed to light may also be affected. The skin first appears bright red and there is a distinct line of demarcation from the normal skin. Later the affected areas of skin become brownish, rough and tend to desquamate. In addition to the back of the neck, the face and backs of hands are often involved and on the face there may be a butterfly distribution across the nose. Note that the lesions are always bilaterally symmetrical. The patient should be questioned regarding his diet and for a history of the following (all commencing with the letter D) – Dyspepsia, Dysphagia, Diarrhoea, Dementia. Dermatitis supplies the fifth D.

Pulsation in the Neck

Pulsation of the carotid arteries may be visible in normal persons but is exaggerated in conditions with a high cardiac output such as thyrotoxicosis and anaemia. Carotid pulsation is also increased in aortic valve incompetence and in coarctation of the aorta. Increased pulsation in the right carotid artery, simulating an aneurysm, is sometimes seen in women with aortic atherosclerosis (unfolding of the aorta) and is due to kinking of the carotid.

Dilated jugular veins can be seen in the supraclavicular regions of the neck in normal persons lying horizontally but disappear as they sit up. To detect abnormal venous dilatation the patient should recline at an angle of 45 degrees and the presence of dilated jugular veins in this position is an important sign of congestive heart failure. It will be noted that the dilated veins pulsate and a quick method of showing that this pulsation is not arterial is to observe its disappearance when applying gentle digital pressure on the jugular vein just above the clavicle. Arterial pulsation is not altered by this manoeuvre. Pulsation does not occur in engorged jugular veins due to constrictive pericarditis, pericardial effusion, obstruction to the superior vena cava in the mediastinum or to constriction of the veins by the deep cervical fascia. Turbulent venous pulsation is indicative of tricuspid incompetence. In differentiating between massive pulmonary embolism and cardiac infarction at the bedside, the presence of early engorgement of the cervical veins would be evidence in favour of pulmonary embolism.

At c sle p n g s d e
P m r y t g
W t b t m s g



Lymph Glands

Tender enlargement of cervical lymph glands may be secondary to a septic focus on the scalp or face or in the mouth nose ear or throat the tonsils are a common focus of infection

Enlarged and tender cervical glands developing after an attack of tonsillitis and associated with splenomegaly fever and malaise suggest the possibility of **glandular fever** (infectious mononucleosis) particularly if there is involvement of other groups of glands Acquired **toxoplasmosis** can closely simulate glandular fever and should be considered when the Paul Bunnell test is negative

Acute leukaemia causes fever prostration ulceration of the mouth and gums purpura and anaemia and later the cervical lymph glands become enlarged

Tender enlargement of cervical and occipital glands may be the first sign of **rubella** (German measles) on the third day the rash appears (see Pashes) and the glands subside as the rash fades

Lymphadenopathy in **scrub typhus** occurs chiefly in the posterior cervical groups but is also present in other parts of

in turn so that it can be decided if the enlargement is diffuse or nodular. A diffuse enlargement unassociated with signs of abnormal thyroid function may be transient or permanent. Transient enlargement occurs in females at times when there is a need for extra iodine, namely at puberty or during menstruation and pregnancy. Permanent and progressive enlargement is liable to occur in regions of the world where the soil and water are deficient in iodine and the condition is known as endemic goitre. Diffuse thyroid enlargement associated with signs and symptoms of hypothyroidism may occur in infants born of goitrous mothers (Swiss cretins) or may signify Hashimoto's disease (lymphadenoid goitre) when occurring in a middle aged woman. Diffuse thyroid enlargement associated with signs and symptoms of hyperthyroidism is diagnostic of Graves's disease (thyrotoxicosis). Nodules in the thyroid are caused by adenomata, either single or multiple, which may or may not give rise to thyrotoxicosis. Three rare conditions involving the thyroid are: acute thyroiditis in which the gland shows all the signs of acute inflammation; Riedel's struma in which the gland becomes stony hard; and carcinoma which should be suspected if an old standing nodule begins to enlarge rapidly or if a part of the gland becomes enlarged, hard and fixed to surrounding structures.

CHEST (THORAX)

The Shape of the Chest

Look for abnormal hollowing above or below the clavicles suggestive of apical tuberculosis or for abnormal fullness above the clavicles suggestive of emphysema

Increased prominence of one side of the chest occurs in scoliosis but if scoliosis is not present make sure that it is not the opposite side of the chest which is abnormal should it be flattened from any cause (e.g. a thickened pleura, pulmonary fibrosis or collapse) the other side will give an impression of being unduly prominent but the flattened side will show diminished respiratory movement. Prominence or fullness of one side of the chest in children may be caused by spontaneous pneumothorax or by a large pleural effusion and the prominent side will show diminished respiratory movement or it may be secondary to gross cardiac enlargement or to pericardial effusion

A rounded hollow at the lower end of the sternum known as funnel chest (trichterbrust) is generally thought to be a congenital deformity and does not cause any symptoms. If the depression in the sternum is deep the heart is displaced to the left

A barrel shaped chest is one which normally assumes the position of full inspiration and the subcostal angle is wide. It is usually indicative of chronic hypertrophic emphysema, a condition brought about by chronic asthma, chronic bronchitis or by bronchiectasis but it should be remembered that thoracic kyphosis may cause the chest to assume a barrel shape. Rickets in infancy may give rise to certain deformities of the chest wall caused by the pull of respiratory muscles on the softened rib, the best known being a girdle like depression passing round the lower ribs anteriorly (Harrison's sulcus or groove), a vertical depression just lateral to the costochondral junctions on each side and an unduly prominent sternum (pigeon breast). In addition the costochondral junctions become enlarged and form a row of knobs on either side of the front of the chest (rickety rosary).

Tietze's disease must be considered in a patient complaining of a painful and tender swelling of one or more costal cartilages in the absence of any abnormality in ribs, heart or lungs. The aetiology of the condition is unknown and it runs a self-limiting course.

A fluctuant swelling over the sternum, over a rib or a costochondral junction on one side, is suggestive of a tuberculous abscess, but the possibility of typhoid osteitis, a late complication of typhoid fever, should be borne in mind.

Pulsation

Cardiac pulsation can usually be observed in the precordial region, but it may not be visible in a proportion of normal persons or when the apex beat of the heart is obscured by emphysematous lung. The apex beat may be more forcible than normal owing to mental excitement or bodily activity in conditions such as *anaemia* and *thyrotoxicosis* where the cardiac output is high, and when the left ventricle is particularly dynamic (aortic valve incompetence).

Arterial pulsation is seen on the chest in only two conditions: (1) an aneurysm of the ascending aorta may produce a swelling showing expansile pulsation to the right of the sternum in the region of the 2nd and 3rd intercostal spaces; (2) the development of a collateral circulation in coarctation of the aorta gives rise to visible pulsation of the intercostal and scapular arteries. These can be seen when the examiner stands behind the patient and asks him to bend forward.

Distended Veins

Visible veins on the chest may be perfectly normal, but if they are distended, there is probably some form of obstruction to the venous blood returning to the heart. This may occur when there is right heart failure or when there is obstruction to either of the venae cavae. When the obstruction is to the superior vena cava or to the innominate veins, the blood in the dilated veins on the chest flows from above downwards; when the obstruction is to the inferior vena cava, the direction of blood flow is from below upwards.

Vascular Lesions

Spider naevi (vascular spiders) have a predilection for the skin over the chest but also occur on the back and the face. They appear as a small central bright red spot from which radiate fine red wavy lines like the legs of a spider. These spider naevi fade on pressure with the finger and are believed to be associated with excess of circulating oestrogens. They occur in pregnancy and may be found in either sex in Cushing's disease, liver cirrhosis and in those receiving a prolonged course of ACTH injections.

Spider naevi have to be differentiated from telangiectases which are networks of dilated capillaries. These are typically seen on the faces of those suffering from rosacea, of those who have had prolonged exposure to wind and sun, and of those who are addicted to alcohol. There is a familial variety known as familial telangiectasia in which epistaxis commonly occurs owing to the presence of telangiectases in the nasal mucosa. These patients often have telangiectases on the ears and on the under surface of the tongue.

EXAMINATION OF THE HEART

The Pulse The pulse should be studied first and thereby early information may be obtained regarding the condition of the heart. Both radial pulses should be felt at the same time observing the rate, rhythm, character and volume.

Rate of the Pulse This is increased by excitement and exercise by most fevers, in heart failure and in paroxysmal tachycardia. The fevers which are not associated with tachycardia and in which there may actually be bradycardia are two bacterial diseases (typhoid and paratyphoid) and seven virus infections (yellow fever, sandfly fever, dengue fever, Q fever, ornithosis, mumps and lymphocytic choriomeningitis).

A slow pulse of 30–40 beats per minute which does not quicken as a result of emotion, exercise or inhalation of amyl nitrite is characteristic of complete heart block (auriculo ventricular block). A pulse of 40–50 beats per minute which doubles in rate on exercise or inhalation of amyl nitrite is due to partial heart block (2:1 block). Heart block, whether partial or complete, is a common toxic effect of digitalis therapy and it may complicate coronary atherosclerosis, Chagas's disease, rheumatic fever and diphtheria. Complete heart block may also occur in association with a ventricular septal defect, with Friedreich's ataxia and with myotonia atrophica.

Patients suffering from heart block are liable to have attacks of loss of consciousness when the heart action undergoes a phase of increased slowing or when the ventricle stops beating. These are known as Stokes-Adams attacks and are associated with stertorous breathing and twitching of the face and limbs. They differ from epilepsy in that there is no aura, the urine is not passed involuntarily nor is the tongue bitten. If the attack is of short duration there may merely be transient loss of consciousness and observers will report that the patient suddenly fainted. Stokes-Adams attacks are dangerous in that the patient may die during the attack as a result of ventricular fibrillation.

Rhythm of the Pulse

One of the commonest types of irregular pulse rhythm is sinus arrhythmia especially in children and is characterised by an increase in pulse rate with inspiration and a decrease with expiration. It is of no clinical significance.

Extrasystoles are premature beats arising from an ectopic focus in auricle or ventricle and occurring just before the normal heart beat would have occurred. They thus replace the normal heart beat and are followed by a compensatory pause which is longer than the normal pause between heart beats. This irregular heart action is reflected in the pulse as a series of normal pulse beats being interrupted suddenly by a premature beat and followed by a compensatory pause. When every alternate heart beat is an ectopic one the pulse has a coupled rhythm and is known as *pulsus bigeminus*. In this condition every two beats of the pulse are followed by a pause and give an impression of recurring in pairs. As with other types of extrasystoles coupled rhythm may occur in healthy persons. Sometimes it is found that these patients are heavy smokers and the irregularity disappears if smoking is given up. It may however occur as a toxic effect of digitalis therapy. If it occurs in the course of such diseases as diphtheria, rheumatic fever or Chagas's disease it may indicate myocardial damage. *Pulsus bigeminus* due to auricular ectopic beats may occur between attacks of auricular paroxysmal tachycardia.

Another type of coupled rhythm occurs in a form of heart block in which every third beat is dropped. The commonest cause in Europe or North America is coronary atherosclerosis but in South America the commonest cause is Chagas's disease. It may also occur as a toxic effect of digitalis therapy or during the course of an attack of diphtheria or rheumatic fever. Any difficulty in differentiating these two forms of coupled rhythm can be resolved by the electrocardiogram.

Character of the Pulse A water hammer pulse sometimes called a collapsing or Corrigan pulse is one with a sharp rise and fall and is characteristic of aortic incompetence.

A pulse which is small and sustained known as a plateau or slow rising pulse is found in aortic stenosis.

If aortic incompetence and stenosis occur in the same heart each pulse wave gives two impulses instead of one (pulsus bisferiens) and the pulse rate appears to be doubled. A pulse with a similar double character may occur in a quite different circumstance namely when there is peripheral vascular failure as may occur in shock and in infections such as typhoid and malaria particularly if associated with dehydration. This is known as a dicrotic pulse.

Volume of the Pulse When a pulse beat of good volume alternates with one of small volume the condition is known as pulsus alternans. It is of no prognostic importance when it occurs during an attack of paroxysmal tachycardia but when not associated with a very rapid pulse it is indicative of toxic carditis or of left ventricular failure.

In constrictive pericarditis and sometimes in pericardial effusion the pulse becomes weaker on inspiration and this variation in volume according to phases of respiration is known as pulsus paradoxus.

An inequality in volume on comparing the two radial pulses calls for further investigation into the possibility of an abnormality in the aorta or in one of the subclavian arteries. For example weakness of the right radial pulse is associated with an aneurysm of the ascending aorta or with a cervical rib pressing on the right subclavian artery. Weakness of the left radial pulse may occur in aneurysm of the aortic arch in coarctation of the aorta and when a cervical rib presses on the left subclavian artery.

In auricular fibrillation the pulse is irregular in time and in force the result being a gross irregularity which is quite characteristic. Occasionally this irregularity may be simulated by frequent extrasystoles and in such cases it will be found that the irregularity of auricular fibrillation becomes more pronounced if the heart rate is increased by the inhalation of amyl nitrite or by exercise while the irregularity of extrasystoles is abolished. It is particularly important for the practitioner to recognise auricular fibrillation for it is indicative of myocardial damage occurring as a complication of such conditions as hypertension, coronary artery disease, mitral stenosis, thyrotoxicosis or Chagas's disease.

The Vessel Wall The walls of the radial artery can be palpated by compressing the artery with the examining finger and rolling it against the underlying bone but it must be remembered that a finding of hardness and tortuosity in the radial or brachial arteries does not signify that the coronary arteries are diseased or that the blood pressure is raised

A particular form of arteriosclerosis in which the media becomes irregularly calcified is known as Monckeberg's sclerosis and can easily be diagnosed by palpating the radial artery. The wall feels as if it had been ringed like a trachea with bands or hoops

Palpating the Praecordium

The apex beat is defined as that point of the anterior chest wall furthest outwards and downwards at which the heart beat can be distinctly palpated. In normal subjects it is situated in the fifth left intercostal space just within the mid clavicular line – a vertical line crossing the mid point between the tip of the acromion and the middle of the suprasternal notch. If the apex beat is in the 5th space but is outside the mid clavicular line it is important to exclude the possibility that the heart has been pushed or pulled to the left as a result of some pulmonary abnormality. An apex beat in the 6th space outside the mid clavicular line signifies an enlarged heart – a particularly likely cause being enlargement of the left ventricle. With the palm of the right hand covering the apex beat make an estimate of the type of cardiac impulse – a sharp slapping impulse occurs with mitral stenosis whereas a heaving impulse in which the hand is raised from the chest wall is characteristic of aortic valve disease. With the hand in this position and then placed over the sternum at different levels test for cardiac thrills – a sense of fine tremulous vibration caused by blood flowing through the heart in a turbulent fashion owing to the presence of unnatural structural impediments within it. A systolic thrill felt best in the 3rd interspace at the left border of the sternum suggests a ventricular septal defect at the pulmonary base suggests pulmonary stenosis and over the aortic base is indicative of aortic stenosis. A diastolic thrill is felt over the apex beat in mitral stenosis and over the lower end of the sternum in aortic insufficiency (accentuated with the patient sitting up and leaning forward) and a thrill lasting

through systole and diastole felt over the pulmonary base accompanies the classical machinery murmur of a patent ductus arteriosus

Auscultation

On auscultating the heart the examiner should pay as much attention to the heart sounds as to any murmurs which occur between them. The 1st sound at the apex is accentuated and has a slapping quality in mitral stenosis. The 2nd sound may be accentuated in the aortic area in hypertension and in the pulmonary area a loud 2nd sound can occur normally or may indicate an increased pressure in the pulmonary circulation (mitral stenosis patent ductus atrial septal defect ventricular septal defect Eisenmenger complex pulmonary artery disease and pulmonary artery embolism) but it is more usual for the 2nd sound to be split and for the second of the split sounds to be accentuated. At the mitral area (apex beat) the first sound may be split in healthy persons and is best heard with the patient sitting or standing up or it may occur when there is impairment of the conducting mechanism of the heart.

Triple Rhythm

A third or diastolic heart sound occurring soon after the 2nd sound may be heard sometimes in normal children and young adults but when it occurs in a patient with cardiovascular disease whose heart rate is over 100 beats per minute it is called a protodiastolic gallop rhythm. It is usually an accompaniment of a failing right ventricle in conditions with pulmonary hypertension but it can be associated with congestive cardiac failure in which both the right and left ventricles are involved i.e. ischaemia thyrotoxicosis anaemia and the late stage of hypertensive failure. Auricular fibrillation is often associated.

A fourth or presystolic heart sound can occur just before the 1st sound in left ventricular failure or when the left ventricle is about to fail particularly in hypertensive heart disease and this is known as a presystolic gallop rhythm. It is never heard if the auricle is fibrillating as auricular contraction is necessary for its manufacture (and there is no auricular contraction during fibrillation).

The Opening Snap This extra heart sound is heard in mitral stenosis just after the 2nd sound and is associated with the opening of the mitral valve

Heart Murmurs

In attempting to interpret cardiac murmurs it will be found advantageous to press lightly on the chest to use a diaphragm chest piece for high pitched sounds such as an aortic diastolic murmur and to use a bell chest piece for low pitched sounds such as a mitral diastolic murmur

Systolic Murmurs A faulty interpretation of mitral systolic murmurs has in the past been responsible for much unnecessary invalidism among children (iatrogenic heart disease) but modern teaching has done much to remedy matters. It is important for the practitioner to appreciate that the vast majority of systolic murmurs heard in the mitral area are functional (innocent) and of no clinical importance. These murmurs are usually soft and blowing in character, are not localised (i.e. they can be traced out towards the left axilla or up towards the pulmonary area), alter in quality or disappear when the patient stands up or takes a deep breath, tend to occur in mid or late systole and are not associated with a palpable thrill or with any other evidence of organic heart disease. This latter point is helpful in correctly interpreting the minority of functional murmurs which happen to be loud and unaltered by posture. Functional murmurs are particularly common in anaemic patients when they go by the name of haemic murmurs. The above remarks also apply to innocent systolic murmurs heard at the pulmonary area or to a systolic murmur which is present at both the mitral and pulmonary areas.

An organic mitral systolic murmur due to disease or deformity of the mitral valve is rough or harsh in character, is localised to the mitral area, commences early in systole, is scarcely altered by posture or by deep respiration and may be associated with a palpable systolic thrill. The commonest cause is rheumatic heart disease, the mitral incompetence being due to shortening and thickening of the chordae tendineae and the systolic murmur is often associated with other murmurs.

of organic heart disease such as a mitral diastolic murmur (mitral stenosis) or a diastolic murmur at the lower left border of the sternum (aortic incompetence)

In the tropics a finding of a loud apical systolic murmur in the absence of any signs of mitral stenosis or aortic valve disease with or without signs of congestive heart failure is suggestive of endomyocardial fibrosis a heart disease of unknown aetiology in which fibrous tissue develops on the endocardial surface of the ventricles. The mitral valve is invariably distorted and incompetent and the tricuspid valve is sometimes involved in addition

A systolic murmur heard best at the left border of the sternum in the 3rd and 4th spaces, often associated with a systolic thrill is suggestive of a ventricular septal defect. The 2nd sound at the pulmonary area will be accentuated or split. There may be evidence of auriculo ventricular block and often other congenital cardiac defects are present

A systolic murmur heard best at the pulmonary area may be due to an atrial septal defect or to pulmonary stenosis. The former is likely if there is no thrill and if the 2nd sound at the pulmonary area is accentuated or split whereas pulmonary stenosis is indicated if there is a systolic thrill and the pulmonary 2nd sound is neither accentuated nor split. In addition half the cases of atrial septal defect have a diastolic murmur at the pulmonary area due to functional incompetence of the pulmonary valve resulting from enlargement of the right ventricle (Graham Steell murmur)

A rough systolic murmur which lasts through systole reaches its maximum intensity at the end of systole wanes in diastole and is often accompanied by a systolic thrill, is the so called *machinery murmur* of a patent ductus arteriosus. The 2nd sound at the pulmonary area is split and the second element of the split sound (the pulmonary element) is accentuated

A long rough systolic murmur at the aortic area conducted up into the neck is indicative of aortic stenosis. There is a palpable thrill and this is more readily felt if the patient sits up leans forward and breathes out. Sometimes the murmur can be traced across the chest as far as the mitral area

Diastolic Murmurs A diastolic murmur of mitral stenosis is best heard at the mitral area with the patient reclining and inclined to the left. It is low pitched has a rumbling quality and can be accentuated by increasing the heart rate. It does not follow immediately after the 2nd sound but is separated from it by a gap and it may have a presystolic accentuation. It can be diagnosed with certainty if the 1st heart sound has a slapping quality and if there is an opening snap immediately after the 2nd heart sound but it should be remembered that neither of these signs will be present if mitral incompetence is present.

A diastolic murmur of similar character heard at the mitral area may be due to gross aortic incompetence (Austin Flint murmur) but should not be confused with the murmur of mitral stenosis as there is no slapping first sound no opening snap the apex beat is displaced outwards and downwards the cardiac impulse is heaving in type and there is a water hammer pulse.

The diastolic murmur at the pulmonary area known as the Graham Steell murmur has already been described.

The characteristic murmur of aortic incompetence is a soft murmur of high pitch which is continuous with the 2nd heart sound and is heard best down the left border of the sternum with the patient sitting up leaning forward and holding his breath after having exhaled. It has a diminuendo quality in that its intensity is greatest at first and then gradually diminishes.

Pericardial Friction This sound is usually first heard in the 2nd or 3rd interspaces close to the sternal border but its position may vary from day to day. It is evidence of pericarditis or of a pericardial effusion which is not large enough to separate the visceral and parietal layers of the pericardium. Pericardial friction can be likened to the sound obtained by rubbing together two pieces of leather and can be differentiated from pleural friction by the fact that the sounds continue when the patient holds his breath.

Heart Failure

Of the many causes of heart failure there are a few which are of particular interest to the practitioner in the tropics and these will be described.

Severe Anaemia This gives rise to breathlessness lassitude and a consciousness of rapid heart action (palpitations) on making slight muscular effort. A haemic murmur may be present and the conjunctivae are pale. Angina of effort may occur and is due to anoxia of the heart muscle owing to the poor oxygen carrying power of the blood. If the left ventricle fails before the right ventricle failure will be ushered in by an attack of paroxysmal dyspnoea due to acute pulmonary oedema. This is particularly likely to occur if the circulation is overloaded by blood transfusion or saline infusion. If the right ventricle fails before the left signs of right heart failure will be present such as raised jugular venous pressure hepatic engorgement ascites and dependent oedema. If the two ventricles fail at the same time there will be in addition to the signs of right heart failure evidence of left heart failure in the form of orthopnoea and pulmonary crepitations.

Beriberi Cardiac failure in beriberi usually presents with the signs of right heart failure (wet beriberi) and the patient should be examined for evidence of peripheral neuropathy. Wernicke's encephalopathy and Korsakow's psychosis. Peripheral neuropathy is shown by weakness and sensory disturbance of the limbs absent tendon reflexes and tender calf muscles. Wernicke's encephalopathy is associated with abnormal eye signs such as nystagmus ptosis impaired eye movements (ocular palsies) and strabismus. Korsakow's psychosis is characterised by mental changes such as apprehension apathy or excitement loss of memory for recent events disorientation in time and space hallucinations and confabulation.

Sometimes heart failure in beriberi develops suddenly especially if the patient has been attempting heavy manual work or is anaemic and in such cases the left ventricle is involved as well as the right giving rise to the acute dyspnoea of pulmonary oedema in addition to the dependent oedema and raised jugular venous pressure of right ventricular failure. The picture is likely to be complicated by some or all of the signs of neuropathy encephalopathy or psychosis as mentioned above.

Endomyocardial Fibrosis Failure may occur in the right ventricle or in both ventricles. Some patients have no evidence

of valvular incompetence but the majority have evidence of mitral incompetence or of combined mitral and tricuspid incompetence

Pulmonary Hypertension Chronic hypertensive pulmonary heart disease may occur as a complication of schistosomiasis when large numbers of bilharzial ova are deposited in and around the walls of the pulmonary arterioles giving rise to pulmonary endarteritis. The right ventricle enlarges and eventually fails. If the patient has resided in a region where schistosomiasis is endemic enquiry should be made for a history of the Katayama syndrome (see p. 61) and the patient should be questioned on terminal haematuria and chronic dysentery. The urine and stool should be examined for bilharzial ova.

Chagas's Disease (South American Trypanosomiasis) In this disease there is diffuse myocardial damage and cardiac failure is not confined to any one side of the heart. Failure is preceded by cardiac arrhythmia such as extrasystoles, partial heart block, complete heart block or auricular fibrillation. Suspicion of Chagas's disease will be strengthened if there is a history of a previous pyrexial illness associated with unilateral periorbital oedema and regional lymphadenitis. Enquiry should be made regarding any difficulty in swallowing or in defaecating for megaoesophagus and megacolon are common complications of this disease.

Toxoplasmosis This should be suspected in a patient of whatever age who presents with heart enlargement or heart failure of unknown aetiology, i.e. in the absence of signs of valvular disease, severe anaemia, thyrotoxicosis, hypertension, beriberi, etc. Both sides of the heart are enlarged, the apex beat being displaced outwards and downwards and cardiac arrhythmia may be present. Failure may occur in the right ventricle, the left ventricle or in both. Sometimes the patient reports with a pyrexial illness simulating glandular fever. Proof of the diagnosis rests on certain specialised tests on the serum (see p. 152).

EXAMINATION OF THE LUNGS

Respiration

Respiratory expansion should be equal on the two sides of the chest and should be confirmed by inspection and palpation. Difficulty in inspiration is associated with any form of obstruction to the larynx or trachea and may give rise to stridor e.g. laryngeal spasm in tetany, laryngeal obstruction in diphtheria, laryngeal oedema in leprosy and angioneurotic oedema, bilateral laryngeal paralysis in poliomyelitis. Difficulty in expiration accompanied by wheezing is a manifestation of a pulmonary abnormality such as asthma or emphysema but it should be remembered that an attack of dyspnoea with wheezing respirations occurring at night may have an entirely different aetiology and be due to left ventricular failure. This is known as cardiac or paroxysmal cardiac dyspnoea and if untreated may go on to pulmonary oedema (see p. 62).

Air hunger affects both inspiration and expiration and occurs in acidaemia (diabetic coma, uraemia).

Cheyne Stokes breathing is a peculiar type of respiration in which successive respirations become deeper and deeper until they reach a maximum and then decrease to a minimum only so start the cycle again following a period of apnoea. This may occur in healthy children during sleep but in adults it may be a sign of myocardial damage, uraemia, increased intracranial pressure or of morphine poisoning.

Cough

Dry Cough A dry unproductive cough is probably due to **pharyngitis** if the pharynx is inflamed and **laryngitis** is the most likely cause if there is an associated huskiness of the voice. Tuberculous laryngitis must be suspected if the dry cough (which may also be painful) does not clear up in a reasonably short time. **Acute tracheobronchitis** may occur as an isolated upper respiratory tract infection but is more common as an accompaniment of a wide range of virus and bacterial

diseases it later tends to become productive as the inflammation descends the bronchial tree (**bronchitis**). Attacks of bronchitis in asthmatic patients (**asthmatic bronchitis**) are always associated with wheezing and prolonged expiration. Continual ineffective coughing is characteristic of **chronic hypertrophic emphysema**. The cough in the early stages of pulmonary tuberculosis is a dry one and tuberculosis should always be suspected in any patient under 40 who has had a dry cough for more than four weeks. Similarly **bronchial carcinoma** should be suspected if the patient is over 40 but the possibility of lung cancer occurring in those under 40 and of lung tuberculosis occurring in those over 40 should not be overlooked. Pressure on one or other recurrent laryngeal nerve in the mediastinum such as might occur from an aortic aneurysm, enlarged lymph glands or from malignant disease causes a cough to have a resonant quality owing to vocal cord paralysis. This type of cough is called **brassy** or **bovine**. Vocal cord damage in lepromatous leprosy can give rise to this type of cough.

In regions where worm infestations are endemic a febrile illness in which there is a dry cough with or without asthma may be an allergic manifestation occurring in the early stages of the life cycle of schistosomes (**Katayama syndrome**), round worms (**Löffler's syndrome**) and filarial worms (**tropical eosinophilia**). Clinical examination of these patients may reveal lymphadenopathy and hepatosplenomegaly and blood examination always reveals an absolute eosinophilia.

The cough in **pertussis** is quite characteristic, consisting of a number of rapidly repeated expiratory coughs followed by a long drawn inspiration as the glottis is partially closed by spasm; the inrush of air to the lungs produces a high pitched sound known as the **whoop**.

The various forms of **pneumonia** such as lobar pneumonia, virus pneumonia and Q fever have a dry cough at first which is painful if the pleura is inflamed and later the cough becomes productive.

A short dry cough in a healthy adult aggravated when he is embarrassed is typical of a **nervous cough** and a loud barking cough present when the patient knows he is being observed is a **hysterical cough**.

Productive Cough A chronic cough worse in the mornings on rising and productive of copious foul smelling sputum is typical of **bronchiectasis** and the patient is likely to have **clubbing** of the fingers and toes. The sputum when allowed to stand in a conical glass settles in three layers – an upper layer of frothy mucus a middle layer of cloudy fluid and an opaque layer consisting of pus and debris

A **lung abscess** may suddenly rupture into a bronchus or bronchiole producing a large quantity of purulent sputum after which there is temporary or permanent improvement. The pus should be sent away for culture. Should such an event take place in amoebiasis as a result of a liver abscess rupturing through the diaphragm the sputum is the colour of anchovy sauce but it may be yellow green in colour if contaminated with bile

Bronchopneumonia may develop from an attack of bronchitis especially in those who are undernourished or otherwise debilitated. It may result from the inhalation of material from the nose or throat or it may complicate specific fevers such as typhoid typhus measles etc. The sputum is mucopurulent and greenish in colour. Tuberculosis in the tropics often presents as bronchopneumonia

In **lobar pneumonia** the sputum is rusty and viscid. It clings firmly to the bottom of the sputum mug and is not dislodged when the latter is inverted

In **pneumonic plague** the patient is febrile dyspnoeic has an anxious facial expression and expectorates profuse and watery blood tinged sputum. This clinical picture can be closely simulated by an attack of left ventricular failure complicating aortic valve disease or severe hypertension. The patient wakes up in the night struggling for breath and expectorating frothy watery fluid which may be blood tinged. There is no fever however the heart will have a presystolic gallop rhythm (see p 54) and there will be other evidence of cardiovascular disease

A history of chronic cough worse in the mornings the sputum being gelatinous in consistency and rusty brown in colour is typical of **paragonimiasis** and microscopical examination of the sputum for ova confirms the diagnosis. Frank haemoptysis

may occur. Although the lungs are the commonest site for encystment of the worms, other regions of the body may also be involved, such as pleura, brain and abdomen, causing symptoms such as chest pain, epilepsy, visual disturbance, weakness of limbs and abdominal pain. The stools should be examined for eggs of the parasite.

A productive cough occurs in the later stage of bronchial carcinoma, and the sputum sometimes, although by no means always, resembles red currant jelly or prune juice.

The sputum of coalminers or of those who live in industrial cities in which coal is used as a fuel, has varying degrees of blackness.

In hydatid disease of the lung, if the cyst ruptures into a bronchus, clear fluid with a salty taste wells up into the mouth; it should be centrifuged and examined for hooklets.

In pulmonary actinomycosis, purulent sputum may be expectorated in which small yellow sulphur granules, the size of a pin's head, may be seen with the naked eye.

Palpation of the Chest Palpation enables the examiner to assess chest expansion, to discover if the trachea is central and to test for vocal fremitus. Any difference in chest expansion on the two sides can be confirmed by placing the hands on either side of the chest, and the position of the trachea can be ascertained by placing the index finger in the suprasternal notch. If the trachea is deviated to one side, it is suggestive of mediastinal shift due to some factor such as pleural effusion or pneumothorax pushing the mediastinum, or to some factor such as pulmonary fibrosis or collapse pulling the mediastinum. A barrel-shaped chest with a wide subcostal angle and poor respiratory movement is characteristic of chronic hypertrophic vesicular (large lung) emphysema.

By placing the palm of the hand on the chest and asking the patient to repeat a number such as ninety-nine, the vocal fremitus can be palpated. It is increased when the lung is consolidated and is diminished or absent when the lung is emphysematous or when it is separated from the chest wall by fluid (pleural effusion), by air (pneumothorax) or by thickened pleura.

THE SPUTUM

The practitioner should never miss an opportunity to look at a patient's sputum for useful information can be obtained by so doing and when required a specimen can be sent away for microscopical examination and culture. This is particularly important in the tropics in view of the high incidence of pulmonary tuberculosis and microscopical examination will also be required to confirm a clinical diagnosis of paragonimiasis or of pneumonic plague.

For the naked eye appearance of various types of sputum see Productive Cough p. 62.

EXAMINATION OF THE BREASTS

Useful information is sometimes gained from an examination of the female breast. Pregnancy is associated with enlargement of the breasts and a brown pigmentation develops in the area immediately around the nipple together with small rounded swellings known as Montgomery's tubercles.

Chronic mastitis gives rise to multiple small rounded elastic swellings in one or both breasts, tenderness sometimes being present, or there may be a single firm painful nodular lump which becomes less noticeable when palpated with the flat of the hand.

An innocent tumour is painless, is freely movable within the breast tissue, does not become attached to the skin, and does not cause any retraction of the nipple nor any enlargement of axillary lymph glands.

A malignant tumour is also painless but is stony hard, is firmly attached to the rest of the breast tissue, often feels irregular, becomes adherent to the skin, frequently leads to retraction of the nipple, is easily felt by the flat of the hand, causes enlargement of axillary gland, and later leads to shrinking, puckering and ulceration of the affected breast. It should be remembered that the male breast may become the seat of malignant disease and the development of a hard painless lump beneath one nipple together with enlargement of axillary glands should arouse immediate suspicion.

Bilateral enlargement of the male breast (gynaecomastia) is usually a manifestation of oestrogen androgen imbalance. Increased circulating oestrogen occurs in fibrosis of the liver, in adrenal cortical tumour, and during stilboestrol therapy for carcinoma of the prostate. Decreased circulating androgen occurs in a group of disorders associated with bilateral testicular atrophy, of which the most important in the tropics is lepromatous leprosy. Castration is a rare cause. Temporary gynaecomastia has been recorded in prisoners of war when feeding well after a prolonged period of starvation. Tenderness and slight swelling of the breasts may occur as a transient phenomenon in infants (mastitis of the newborn) and in boys at puberty (mastitis of puberty).

THE BACK

The Spine With the patient standing up look for any abnormal curvature of the spine feel for any deformity or tenderness and ask the patient to carry out movements which will test the spine's mobility

Lateral curvature (scoliosis) is commonly **postural** either because of faulty habits of posture or to compensate for shortening of one leg Weakened muscles from poliomyelitis malnutrition or prolonged illness may sometimes play a contributory role

Posterior curvature (**kyphosis**) usually present in the thoracic region may be **postural** but often is due to underlying bone disease **Tuberculous caries** commonly occurs in childhood and is associated with palpable deformity of one or more vertebrae with tenderness of the affected vertebrae A cold abscess may be present in addition An old **crush fracture** will be suspected if there is a non tender palpable deformity of one vertebra and a history of previous injury **Decalcification** of the spine may arise from osteoporosis (atrophy of the organic matrix) or from osteomalacia (defective calcium metabolism) Kyphosis due to osteoporosis is classically seen in old age the bone changes being secondary to lack of sex hormones while kyphosis due to osteomalacia is typically seen in conditions where there is decreased absorption of calcium in the gut This may be secondary to lack of vitamin D (*rickets*) or to intestinal changes (*idiopathic steatorrhoea*) but there is a group of rarer conditions in which the calcium deficiency is not due to faulty absorption but to excessive loss in the urine (*renal rickets* *osteitis fibrosa cystica* *Fanconi syndrome* and *idiopathic hypercalcuria*) **Osteitis deformans** (Paget's disease) occurs after middle age and is associated with bowing of the legs and increase in the size of the head **Ankylosing spondylitis** (*spondylitis ankylopoietica*) commences in young adults and leads to a rigid spine with a diffuse kyphosis When the *cervical vertebrae* are also involved the head becomes fixed in a forward position so that the unfortunate patient when standing can see only the ground at his feet Thoracic kyphoscoliosis occurs in acromegaly (see p 98)

Anterior curvature (lordosis) is confined to the lumbar and lower dorsal regions and is an exaggeration of the natural hollow of the back. It may be compensatory in an attempt to counterbalance a kyphosis higher up the spine or disease of the hips lower down, or it may be due to muscular weakness as occurs typically in children suffering from muscular dystrophy.

Examination of an infant's back may reveal a cystic swelling over the spine – **spina bifida cystica**. This is called a **meningocele** if it contains only the meninges and cerebrospinal fluid and is called a **meningomyelocele** if it contains, in addition, the spinal cord or the nerves of the cauda equina. In **spina bifida occulta**, some of the vertebral arches are absent but there is no protrusion of the meninges or spinal contents; the overlying skin is thick and puckered and may show an excessive growth of coarse hair.

A dimple or sinus is occasionally seen over the sacro-coccygeal region and is due to the remnant of the embryological structure known as the **neurenteric canal**, causing traction on the skin at this point.

Of the various congenital tumours situated in the sacro-coccygeal region, a **dermoid cyst** is the least rare. Such a cyst is adherent to deeper tissues but not to the skin, and as it is lined with epithelium, it contains hairs and sebaceous material.

The Scapulae – Winging of the scapulae is a classical sign of muscular dystrophy and occurs in the **pseudohypertrophic** type and in the **scapulo-humeral** type; the common factor being bilateral paralysis of **serratus anterior**. It occurs unilaterally when the long thoracic nerve is damaged.

There is a rare congenital deformity in which one scapula is in a high position with the lower angle turned towards the spine. This is known as **Sprengel's deformity** (**Sprengel's shoulder**) and the patient cannot raise the arm fully on the affected side.

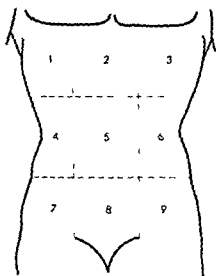
Fluctuant Swellings in the Back – A fluctuant swelling in the soft tissues of the back, non-tender and unassociated with constitutional disturbance or the usual signs of sepsis, is suggestive of a **tuberculous abscess** secondary to tuberculous infection of the spine or ribs.

A rare cause of a fluctuant swelling in the back is thoracic actinomycosis. It differs from the cold abscess of tuberculosis in that it is likely to be painful and to cause redness and oedema of the overlying skin.

An ill febrile patient reporting with a tender swelling in one loin is probably suffering from a perinephric (perirenal) abscess.

Nodules Palpation along the iliac crests in a patient complaining of pruritus of thighs, buttocks and back principally may reveal the typical subcutaneous nodules of onchocerciasis. In the early stages these are the size of a lentil and their stony hardness can be observed when they are palpated against the iliac bone. They slowly enlarge and years later if the patient is untreated they may become visible as well as palpable.

EXAMINATION OF THE ABDOMEN



- 1 Right hypochondrium
- 2 Epigastrium
- 3 Left hypochondrium
- 4 Right lumbar region
- 5 Umbilical region
- 6 Left lumbar region
- 7 Right iliac fossa
- 8 Hypogastrium
- 9 Left iliac fossa

Inspection The urinary bladder having been emptied the abdomen is examined while the patient is recumbent. Note any abnormality of contour such as a localised bulging as can occur over an enlarged liver or spleen. Note also in which zone of the abdomen the swelling appears (see Diagram) and also if it moves on respiration.

A uniform distension of the abdomen with fullness in each flank together with a transversely stretched umbilicus and a diminution in abdominal respiratory movements are important signs of ascites. Distension in the central portion of the abdomen occurs in the sprue syndrome owing to gas produced by fermentation in the small intestine. Generalised gaseous distension of the abdomen occurs in acute intestinal obstruction and in paralytic ileus.

In a female swelling of the lower abdomen without fullness in the flanks may be due to a uterus enlarged by pregnancy or fibroids or to a cyst or tumour of the ovary bulging out of the pelvis. In such cases the umbilicus is shifted upwards.

The normal rise of the abdomen on inspiration and fall on expiration may be absent or reversed on one side if the diaphragm on that side is paralysed this may prove a valuable clue to the presence of a sub diaphragmatic abscess particularly to an amoebic abscess of the liver Peritonitis is another condition which depresses abdominal respiratory movements

Visible pulsation of the abdominal aorta occurs normally particularly in thin patients but the presence of dilated superficial veins on the anterior abdominal wall the blood flowing in an upward direction occurs when the inferior vena cava is obstructed

When examining a patient with suspected gastro intestinal disease the abdomen should be closely inspected for visible peristaltic waves sometimes these can be stimulated by flicking the abdomen with a finger Their presence indicates a very thin abdominal wall as in the sprue syndrome or may be a sign of pyloric or intestinal obstruction

Transverse white scars in the skin of the abdomen called lineae albicantes indicate that the abdomen has been considerably swollen at some time in the past and an obese abdomen with purple scars on it (lineae atrophicae) is typical of Cushing's syndrome A line of pigmentation stretching from the symphysis pubis to beyond the umbilicus appears in dark complexioned women at about the fourth month of pregnancy and is called linea nigra

An umbilical hernia or a post operative incisional hernia will become prominent when the patient is asked to raise his shoulders from the couch without the aid of his arms

Discharging sinuses in the right iliac fossa which developed after the appendix had been removed for appendicitis indicate that the true diagnosis had been missed at the time of the operation In such a case it is important to exclude psoas abscess ileocaecal tuberculosis amoebic typhlitis and caecal actinomycosis Examination of the pus is an essential investigation for inspection will reveal the characteristic sulphur granules in actinomycosis and laboratory examination will show trophozoites of *Entamoeba histolytica* in amoebiasis and tubercle bacilli in tuberculosis The sulphur granules should be examined microscopically for the delicate branching filaments of *Actinomyces bovis*

The abdomen should be carefully inspected for the presence of a rash particularly in order to avoid missing the rash of typhoid fever in a patient being examined for P.U.O. (pyrexia of undetermined origin) – see Rashes p 135

Palpation The abdomen should be systematically palpated in order to assess the resistance of the abdominal muscles to find any evidence of tenderness and to discover any palpable masses

Rigidity of the abdominal muscles unless occurring in a nervous or ticklish patient is suggestive of peritonitis

A thin patient whose abdomen feels doughy requires investigation for the sprue syndrome (tropical sprue idiopathic steatorrhoea coeliac disease). He should be questioned regarding the passage of copious pale foul smelling stools and should be asked if he has suffered from sore mouth or from tetany. An earthy colouration of the skin and a smooth red (raw beef) tongue are helpful signs in diagnosis. If he has a surgical scar on the upper abdomen and the history shows that the steatorrhoea commenced after the operation he is probably suffering from some form of intestinal by pass which is responsible for his malabsorption syndrome such as gastro colic fistula or jejuno colic fistula.

The Epigastrium Tenderness just below the xiphisternum in a patient complaining of recurrent epigastric pain suggests the presence of a gastric ulcer and tenderness at a point half way between xiphisternum and umbilicus about 2 cm. to the right of the mid line is suggestive of a duodenal ulcer.

A distended gall bladder forms a smooth pear shaped palpable swelling situated just beyond the outer edge of the right rectus muscle. It descends with respiration and can be moved from side to side. It is present in some cases of biliary obstruction and may provide strong supporting evidence for a diagnosis of carcinoma of the head of the pancreas. Such a swelling may be simulated by a Riedel's lobe of the liver but this cannot be moved from side to side. A tender gall bladder is found in cholecystitis and has to be distinguished from hepatic tenderness due to an amoebic abscess in the antero inferior portion of the right lobe of the liver. A helpful point in differ

entiating these two conditions is that subcostal tenderness is strictly localised in cholecystitis but is more diffuse in amoebic abscess

A hard and irregular mass palpated in the lower part of the epigastrium is a sign of carcinoma of the stomach but it should be possible to diagnose this condition before the stage is reached when a mass can be palpated on abdominal examination. The mass is movable at first and is tender on pressure but it later becomes fixed. Visible peristaltic waves passing from left to right occur when there is obstruction at the pylorus. This sign is often absent however but can usually be demonstrated by getting the patient to drink a glass of soda water.

The Right Hypochondrium This region of the abdomen is palpated for evidence of liver enlargement bearing in mind the fact that in infants and young children the lower edge of the liver is normally palpable 1–1½ cm below the right costal margin in the mid clavicular line on full inspiration. When examining a patient who has a palpable liver find the upper level of liver dullness by percussing the front of the chest on the right side. Dullness higher than the fifth interspace in the mid clavicular line is abnormal. The lower edge of the liver should be palpated for any tenderness and to learn about the consistence (soft or hard) and the contour (smooth or irregular). Tenderness of the liver edge occurs in certain systemic infections and when the liver is becoming rapidly enlarged as in right sided heart failure.

An important cause of tender liver enlargement in the tropics is **amoebic hepatitis**. The patient complains of fever and pain in the liver region and when the right hypochondrium is palpated the lower edge of the liver will be found to come down below the costal margin on inspiration. Careful percussion of the chest will reveal that the liver is enlarging upwards as well as downwards. If treatment is withheld at this stage the rapidly growing colonies of *Entamoeba histolytica* in the liver merge together to form a single amoebic abscess which in turn enlarges. The patient's symptoms become more pronounced as the liver enlarges still further. Irritation of the right hemi diaphragm causes referred pain to the tip of the right shoulder and collapse of the lower lobe of the right lung gives rise to dullness on percussion and absence of breath sounds at the

right base. A small pleural effusion usually develops as well. Jaundice has been known to occur with liver abscess but is very rare. Difficulty may arise in differentiating an amoebic liver abscess from a subphrenic abscess secondary to rupture of an inflamed appendix or of a peptic ulcer but a careful history will help to resolve the problem. For example subphrenic abscess is the probable diagnosis if the patient gives a history of an illness suggestive of acute appendicitis shortly prior to the present illness or there may be a history of previous peptic ulceration with a more recent bout of severe epigastric pain. In either case abdominal examination may be helpful in revealing a tender palpable mass in the region of the caecum or tenderness and rigidity on palpating the epigastrium. Further perforation of any portion of the intestine is liable to cause air to escape into the peritoneal cavity and a subphrenic abscess may therefore be associated with an air bubble which alters its position in relation to the liver depending on the position of the patient. With the patient lying flat the air lies in front of the liver and may give a resonant note on percussion instead of the usual liver dullness. With the patient sitting up and leaning forward the bubble of air insinuates itself between the diaphragm and the liver and percussion at the back of the right chest may reveal an area of resonance between the dull note over the collapsed lower lobe and the dull note over the liver.

Help in diagnosing an amoebic abscess would be obtained if the patient gave a history of previous proved intestinal amoebiasis or if stool examination revealed cysts of *Entamoeba histolytica*. But in the majority of cases neither of these items of information can be elicited. Clinical findings in favour of an intrahepatic abscess would be tenderness on compressing the ribs antero posteriorly tenderness or evidence of bulging at any one particular point on the anterior chest wall (e.g. in the fifth right intercostal space) or tenderness on palpating the lower edge of the liver.

An irregular liver edge is found in four principal conditions. In cirrhosis there is a diffuse distribution of small nodules varying very little in size (hob nail liver). In carcinoma there are hard nodules of various sizes. In tertiary syphilis there may be one or more gummata producing large nodules on the surface of the liver prior to the development of scarring and gross deformity (hepar lobatum) and irregular prominences may be

palpated on the surface of the liver in hepatic actinomycosis closely simulating gummatous or malignant change

Liver enlargement is so commonly encountered in the tropics that it would be advisable at this juncture to consider the possible causes

Causes of Tender Liver Enlargement

(1) **Inflammatory** Amoebic hepatitis amoebic liver abscess pyogenic suppuration

(2) **Systemic infections** Malaria leptospirosis relapsing fever miliary tuberculosis brucellosis infectious hepatitis yellow fever

(3) **Parasitic** During the larval stage of certain helminthic infestations e.g. schistosomiasis (Katayama syndrome) ascariasis (Löffler's syndrome due to *A. lumbricoides* and larval granulomatosis due to *Toxocara canis*) and filariasis (tropical eosinophilia)

(4) **Venous congestion** Congestive heart failure

(5) **Liver poisons** Carbon tetrachloride antimony arsenic and others

Causes of Non tender Liver Enlargement

This occurs when the liver is being infiltrated slowly irrespective of the type of infiltration. The causes are

(1) **Infiltration by parasites** a **Helminths** Schistosomiasis clonorchiasis hydatid disease fascioliasis b **Spirochaetes** Syphilis c **Protozoa** Kala azar trypanosomiasis toxoplasmosis d **Fungi** Histoplasmosis actinomycosis

(2) **Infiltration by fat and lipoids** Fatty infiltration as occurs in nutritional diseases such as kwashiorkor or in chronic poisoning as by alcohol. Lipoid storage diseases (Gaucher's disease Niemann-Pick disease Hand-Schüller-Christian disease gargoylism)



Kel -a

(3) Infiltration by abnormal reticulo-endothelial cells Leuk
aemia Hodgkin's disease disseminated lupus erythematosus

(4) Infiltration by products of red cell destruction Pernicious
anaemia chronic haemolytic anaemia (sickleaemia Mediter
ranean anaemia acholuric familial jaundice icterus gravis
neonatorum)

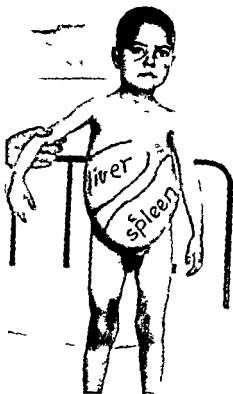
(5) Infiltration by iron Siderosis

(6) Infiltration by amyloid Amyloidosis

(7) Infiltration by glycogen Von Gierke's disease

(8) Infiltration by galactose Galactosaemia

(9) Infiltration by amino-acids Cystine disease (Fonconi syn
drome)



Med to o o
kala o o

(10) **Infiltration by malignant cells** Carcinoma

(11) **Granulomatous infiltration** Sarcoidosis

(12) **Engorgement with blood** Polycythaemia rubra vera, chronic constrictive pericarditis tricuspid stenosis or incompetence

(13) **Infiltration by fibrous tissue (fibrosis)** Haemochromatosis hepatolenticular degeneration (Wilson's disease) and as a late stage of any of the following fatty infiltration infectious hepatitis engorgement with blood deposition of schistosome ova and veno occlusive disease

Left Hypochondrium The spleen is the commonest organ giving rise to a palpable mass in the left hypochondrium. As with palpation of the liver, particular care should be taken to feel for the lower border of the organ while the patient takes long and deep inspirations. In this way the descent of the diaphragm

assists by pushing down the enlarged spleen on to the examiner's fingers. If the lower pole can be felt in this way it can be assumed that the spleen is about twice its normal size. Lesser degrees of enlargement cannot be confirmed clinically. It should be remembered that the spleen may be so grossly enlarged, particularly in kala azar and myeloid leukaemia, that it fills the greater part of the left abdomen and the lower pole may be missed if palpation is commenced too high up.

Causes of Enlargement of the Spleen

In considering the causes of a palpable spleen, many of the causes of hepatomegaly will apply, and the subject can be approached as follows:

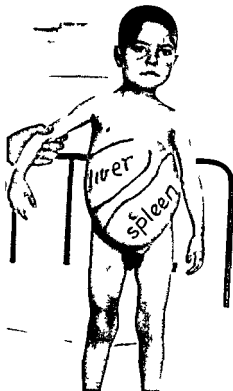
(1) **Systemic infections** a **Protozoal** Malaria, kala azar, trypanosomiasis, toxoplasmosis. b **Spirochaetal** Congenital syphilis, relapsing fever. c **Bacterial** Tuberculosis, typhoid, brucellosis, bacterial endocarditis, septicaemia. d **Rickettsial** Typhus group. e **Viral** Infectious hepatitis, glandular fever.

(2) **Venous back pressure** a Liver fibrosis or cirrhosis. b Cardiac causes: Congestive heart failure, chronic constrictive pericarditis, tricuspid stenosis or incompetence. c Thrombosis of portal or splenic vein.

(3) **Parasitic** During the larval stage of certain helminthic infestations, e.g. schistosomiasis (Katayama syndrome), ascariasis (Löffler's syndrome due to *A. lumbricoides* and larval granulomatosis due to *Toxocara canis*) and filariasis (tropical eosinophilia).

(4) **Certain blood diseases** Pernicious anaemia, iron deficiency anaemia, polycythaemia rubra vera, chronic purpura, haemorrhagica, and chronic haemolytic anaemia (sickle cell, Mediterranean anaemia, acholuric familial jaundice, von Jaksch's anaemia, icterus gravis neonatorum).

(5) **Certain bone disorders** which destroy the marrow and cause the spleen to revert to its foetal function of blood cell manufacture, e.g. myelosclerosis and osteopetrosis (Albers-Schönberg disease).



M d i r a
k a l a a r

(10) **Infiltration by malignant cells** Carcinoma

(11) **Granulomatous infiltration** Sarcoidosis

(12) **Engorgement with blood** Polycythaemia rubra vera chronic constrictive pericarditis tricuspid stenosis or incompetence

(13) **Infiltration by fibrous tissue (fibrosis)** Haemochromatosis hepatolenticular degeneration (Wilson's disease) and as a late stage of any of the following fatty infiltration infectious hepatitis engorgement with blood deposition of schistosome ova and veno occlusive disease

Left Hypochondrium The spleen is the commonest organ giving rise to a palpable mass in the left hypochondrium. As with palpation of the liver, particular care should be taken to feel for the lower border of the organ while the patient takes long and deep inspirations. In this way the descent of the diaphragm

assists by pushing down the enlarged spleen on to the examiner's fingers. If the lower pole can be felt in this way it can be assumed that the spleen is about twice its normal size. Lesser degrees of enlargement cannot be confirmed clinically. It should be remembered that the spleen may be so grossly enlarged particularly in kala azar and myeloid leukaemia that it fills the greater part of the left abdomen and the lower pole may be missed if palpation is commenced too high up.

Causes of Enlargement of the Spleen

In considering the causes of a palpable spleen many of the causes of hepatomegaly will apply and the subject can be approached as follows:

(1) **Systemic infections** a Protozoal Malaria kala azar trypanosomiasis toxoplasmosis b Spirochaetal Congenital syphilis relapsing fever c Bacterial Tuberculosis typhoid brucellosis bacterial endocarditis septicaemia d Rickettsial Typhus group e Viral Infectious hepatitis glandular fever

(2) **Venous back pressure** a Liver fibrosis or cirrhosis b Cardiac causes Congestive heart failure chronic constrictive pericarditis tricuspid stenosis or incompetence c Thrombosis of portal or splenic vein

(3) **Parasitic** During the larval stage of certain helminthic infestations e.g. schistosomiasis (Katayama syndrome) ascariasis (Löfller's syndrome due to *A. lumbricoides* and larval granulomatosis due to *Toxocara canis*) and filariasis (tropical eosinophilia)

(4) **Certain blood diseases** Pernicious anaemia iron deficiency anaemia polycythaemia rubra vera chronic purpura haemorrhagica and chronic haemolytic anaemia (sickle cell Mediterranean anaemia acholuric familial jaundice von Jaksch's anaemia icterus gravis neonatorum)

(5) **Certain bone disorders** which destroy the marrow and cause the spleen to revert to its foetal function of blood cell manufacture e.g. myelofibrosis and osteopetrosis (Albers-Schönberg disease)

- (6) **Infiltration with abnormal reticulo endothelial cells** Leuk aemia Hodgkin s disease and disseminated lupus erythema tosus
- (7) **Certain Rheumatic diseases** Rheumatoid arthritis Still s disease and Felty s syndrome
- (8) **Infiltration with amyloid** Amyloidosis
- (9) **Infiltration with lipid** Lipoid storage diseases (Gaucher s disease Niemann Pick disease Hand Schuller Christian disease gargoylism)
- (10) **Mycotic disease** Histoplasmosis
- (11) **Granulomatous infiltration** Sarcoidosis
- (12) **Cystic change** Hydatid cyst Congenital cyst
- (13) **Abscess**

A tumour of the stomach may be felt in the left hypochondrium and in view of the mobility of the stomach the palpable mass will be found to change position with the patient s change of posture Enlargement of the left lobe of the liver may simulate an enlarged spleen

In protein deficiency the left lobe of the liver may occasionally become enlarged before the right lobe and the liver edge can be palpated on inspiration in the left hypochondrium A possible explanation why fatty infiltration should develop first in the left hepatic lobe is that blood from the small intestine carrying any available lipotropic factors tends to be carried in the portal blood going to the right side of the liver thus depriving the left lobe which receives most of the blood from the spleen and distal colon Another cause of an enlarged left lobe is compensatory hypertrophy

An amoebic abscess of the left lobe of the liver is a rare cause of a palpable mass in the left hypochondrium Concomitant signs would include fever collapse of the lower lobe of the left lung giving dullness to percussion at the left base upward displacement of the heart and pain in the left lower chest referred to the left shoulder

The Umbilical Region The transverse colon and the coils of the small intestine lie immediately under the anterior abdominal wall in this region. The pancreas lies deeper.

A palpable mass in the transverse colon can be moved upwards and downwards and may be faecal, neoplastic or amoebic. A faecal mass (scybalum) is by far the most common and will have disappeared a few hours later or following administration of an enema. Although an amoeboma is less hard and irregular than a malignant growth of the transverse colon, it may be impossible to differentiate these two conditions at the bedside. A course of emetine, however, will cause an amoeboma to disappear.

Tenderness in the umbilical region is commonly found when the small intestine is inflamed, as in food poisoning and typhoid.

A pancreatic cyst may be palpated in the umbilical region as a round, tense swelling deep in the abdomen. It does not move on respiration.

The Right Lumbar Region The ascending colon lies in this region and tenderness may be elicited together with tenderness over other portions of the colon in bacillary or amoebic dysentery and in ulcerative colitis.

A palpable mass arising from the right kidney or adrenal, as opposed to one from bowel or omentum, moves with respiration, but it should be noted that the lower pole of the right kidney may be normally palpable in a small proportion of thin patients. Enlargement of the kidney calls for investigations to exclude various possibilities such as embryoma (Wilms's tumour) in infants, hypernephroma (Grawitz tumour) and polycystic disease in adults, renal tuberculosis and bilateral hydronephrosis or pyelonephritis in any age group.

Rarely the adrenal may be the seat of a palpable tumour and the diagnosis will be simplified by evidence of virilism in a child or of Cushing's syndrome in an adult (see p. 49-72).

The Left Lumbar Region The descending colon is palpable in this region and it may be tender in bacillary or amoebic dysentery, in ulcerative colitis and in acute diverticulitis.

In ulcerative colitis and in spastic colon the descending colon may be palpated as a firm rubbery tube

A palpable mass in the descending colon does not move with respiration but is movable from side to side and the differential diagnosis between scybalum amoeboma and carcinoma has already been described in connection with the transverse colon (see p 81)

The left kidney and adrenal may be enlarged as a result of the pathological changes recorded for the right kidney and adrenal (see p 81) but the left kidney is situated at a slightly higher level than the right and therefore the lower pole is not normally palpable In addition an enlarged left kidney may be mistaken for an enlarged spleen unless attention is paid to the following points

- (1) The inner edge of the spleen is sharp whereas that of the kidney feels rounded
- (2) A notch may be felt on the lower and inner aspect of the spleen but not on the kidney
- (3) The examiner's fingers can be inserted between the posterior aspect of an enlarged spleen and the erector spinae muscle but this is not possible with an enlarged kidney
- (4) The examiner's fingers can be inserted between the anterior aspect of an enlarged kidney and the lowest rib but an enlarged spleen is too close to the ribs to permit this
- (5) Percussion over an enlarged spleen gives a dull note but over an enlarged kidney there is a resonant note owing to overlying colon
- (6) An enlarged kidney can often be moved backwards and forwards between one hand placed in the loin and the other on the anterior abdominal wall This manoeuvre is not possible with an enlarged spleen

The Right Iliac Fossa Tenderness in the right iliac fossa may indicate an inflamed appendix particularly if maximal over McBurney's point and associated with muscle guarding

A crisis in sickle cell anaemia may give rise to marked abdominal tenderness over the site of a mesenteric thrombosis and an attack of malignant tertian malaria may be associated with tenderness in the lower abdomen. Both these conditions may closely simulate an attack of acute appendicitis and the wise practitioner will exclude them before recommending appendicectomy.

A palpable tender mass in the region of the caecum may arise as a complication of acute appendicitis in which case it is due to localised peritonitis with or without abscess formation. The patient is likely to be a child or young adult and there will be a history of a previous attack of fever, vomiting and pain.

Tenderness over the caecum in amoebic typhlitis is not likely to cause muscle spasm and tenderness may sometimes be elicited on palpating other portions of the large intestine particularly the central part of the sigmoid colon in the left iliac fossa (Manson-Bahr's amoebic point). When the caecum is the seat of a chronic amoebic infection it will feel thickened and tender. It can be moved from side to side. Diagnosis will depend on the finding of amoebic cysts in the stool and on demonstrating caecal changes on a barium enema X-ray.

In hyperplastic ileocaecal tuberculosis a firm, tender, fixed, irregular mass will be palpable in the right iliac fossa and its lower border can be better defined than the upper owing to the fact that the disease spreads to the ascending colon but not to the ileum. It may occur at any age and unlike caecal carcinoma is more likely to affect the young than the old. There will be a history of alternating diarrhoea and constipation and search should be carried out for tuberculosis in the lungs. A well equipped laboratory should be able to culture tubercle bacilli from the stools.

In caecal carcinoma the mass is hard rather than firm, may be rounded or irregular, is unlikely to be tender and can be moved from side to side. It usually occurs in patients over 50 and the history is one of increasing weakness and loss of weight with alternating diarrhoea and constipation as a less obvious manifestation.

Caecal actinomycosis gives rise to a mass which is hard, fixed and non-tender and only in the later stages does it discharge

through the anterior abdominal wall via multiple sinuses. It may occur at any age and the history of pain in the right iliac fossa is either of short duration or absent altogether.

Regional enteritis (Crohn's disease) gives rise to a tender fixed palpable mass in the right iliac fossa which is rubbery in consistency. It chiefly affects young adults between 20 and 40 causing abdominal pain, loss of weight and recurrent diarrhoea. Recent observations have brought to light the fact that a similar syndrome may occur as a late result of an infection with the virus of lymphopathia venereum.

Intussusception commences in the region of the right iliac fossa as a tender cylindrical mass which varies in firmness but later may be felt anywhere in the abdomen. It occurs in infants under the age of two years and is accompanied by vomiting, abdominal pain and the passage of blood per rectum. A rare syndrome with buccal pigmentation and intestinal polyposis has been described in children and adults and the patients are liable to recurrent attacks of intussusception.

Acute salpingitis is invariably bilateral and causes tenderness in both iliac fossae close to the midline. There is a history of a purulent vaginal discharge and the swollen and tender Fallopian tubes can be palpated on vaginal examination.

The Left Iliac Fossa Tenderness in the left iliac fossa may occur in sickle cell anaemia, malignant tertian malaria and in acute salpingitis (see R I F p 82-83).

Tenderness of the sigmoid colon is a constant sign in dysentery, ulcerative colitis and in acute diverticulitis.

A firm tender mass is likely to be due to chronic diverticulitis if the patient is over 40. It has to be distinguished from carcinoma, amoeboma, spastic colon and from a scybalum. In carcinoma there is a shorter history of abdominal symptoms, rarely longer than a year, the patient is likely to be cachectic and tenderness may be absent. Further investigations such as sigmoidoscopy and barium enema X-ray are necessary to establish the diagnosis.

An amoeboma may occur at any age and there may or may not be a history of previous dysentery. A finding of cysts or

trophozoites of *Entamoeba histolytica* in the stool will strengthen the diagnosis as will sigmoidoscopic examination. Final proof of the diagnosis rests on the disappearance of the granuloma as a result of a course of emetine.

A spastic colon gives rise to a firm cylindrical tender swelling lying above and parallel to Poupart's ligament. It is a manifestation of hyper excitability of the colon and as it is not associated with any organic changes in the bowel it may be entirely absent at a subsequent abdominal examination. It occurs in patients who are subjected to nervous strains and stresses particularly in the age group 30-50.

A scybalum is non tender and disappears after an enema has been given.

The Hypogastrium An enlarged bladder is tense, tender and fixed in position. It is the commonest palpable swelling in the hypogastrium and this makes it imperative that the doctor should ensure that the patient's bladder is emptied before giving an opinion on the cause of a palpable mass in this region.

The fundus of the gravid uterus can be palpated above the symphysis pubis by the 12th week of pregnancy and it reaches the level of the umbilicus by the 24th week.

The non gravid uterus may be palpated in the hypogastrium when it contains a large fibromyoma. The tumour is of firm consistency and bimanual examination will confirm that it is uterine in origin.

An ovarian cyst presents as a hypogastric tumour with a smooth surface and capable of being moved from side to side. The consistency is tense and a fluid thrill may be elicited. Bimanual examination is necessary to establish the nature of the swelling.

Percussion of the Abdomen

Percussion may be used to confirm enlargement of the liver, spleen or gall bladder. Loss of the normal liver dullness is indicative of air in the peritoneal cavity and if not due to thera

peptic pneumoperitoneum is in keeping with a diagnosis of a perforated peptic ulcer. A tympanitic note is obtained over dilated bowel as for example in gastric dilatation, intestinal obstruction and sprue.

The presence of ascites can be confirmed by percussing one flank when the patient is lying on his back and then percussing the same flank when the patient is turned on to the opposite side. The flank which was dull to percussion in the first position becomes resonant in the second position and this sign is known as shifting dullness. The commonest cause in the tropics is cirrhosis of the liver, the next most common being congestive heart failure. Cardiac causes of ascites, besides heart failure, include constrictive pericarditis and rheumatic disease of the tricuspid valve. Constrictive pericarditis is a tuberculous and not a rheumatic condition and back pressure on the venous return to the heart causes enlargement of the liver with ascites and engorged cervical veins with diminished pulsation. The heart is not enlarged, there are no heart murmurs, a third heart sound is heard at the apex, the lung bases are clear, the pulse is small and paradoxical, auricular fibrillation is often present and the hands are cyanosed and cold.

Tricuspid valve disease is rheumatic in origin and very rarely occurs in the absence of mitral valve disease. It gives rise to dilated cervical veins which show increased pulsation, heart enlargement, heart murmurs which are difficult to distinguish from the murmurs of the associated mitral valve disease, enlargement of the liver with expansile pulsation and ascites. These signs give a false impression that the patient is suffering from right heart failure but this is not so for the patient is practically free from symptoms until heart failure eventually sets in. A finding of ascites in a female over 40 together with a pleural effusion which may be unilateral or bilateral calls for investigations to exclude the presence of an ovarian tumour. The tumour is usually a fibroma and when removed the effusions promptly disappear. This syndrome is known as Meigs's syndrome.

Other causes of ascites include peritonitis (particularly tuberculous), secondary malignant deposits in the peritoneum, obstruction to the portal vein by thrombosis or by pressure from enlarged glands or from tumours of adjacent organs.

If the ascitic fluid is found to be milky (chylous ascites) the cause is obstruction to the thoracic duct or receptaculum chyli from tuberculosis malignancy or Bancroftian filariasis

A large ovarian cyst will not cause confusion with ascites if it is noticed that the dullness to percussion is central rather than lateral and the umbilicus is drawn upwards



Lymphopathia venereum
(climatic bubo)



Gonorrhea

GROINS PERINEUM AND EXTERNAL GENITALIA

These regions should be examined systematically as they are particularly important in tropical practice

Enlarged inguinal lymph glands may be part of a generalised lymphadenopathy in such conditions as the reticuloses glandular fever glandular tuberculosis sarcoidosis secondary syphilis and kala azar. When they are the only lymph glands affected it is necessary to look for skin sepsis in the surrounding area including the buttocks and other possibilities include primary syphilis soft sore lymphopathia venereum (climatic bubo) plague tick typhus and Bancroftian filariasis

In **primary syphilis** the patient is afebrile and a primary sore will be found on the external genitalia. It is easily found in the male as it is invariably situated on the distal part of the penis. In the female it is easily found when situated on the labia



G l m a v e m



S c o d y y w c o d y l a m t a l l

majora or minora but it is often present on the cervix and will not be seen without the aid of a vaginal speculum. The primary chancre is a painless circular or oval granuloma with an eroded or ulcerated surface. It is usually single and feels firm (indurated) when palpated, the firmness being confined to the chancre and not involving the surrounding skin. The glands in the groin are only moderately enlarged, are rubbery, discrete and non-tender, and do not break down.

A **soft sore** (ulcus molle) is much commoner in the tropics than elsewhere and may be confused with primary syphilis as it also causes inguinal lymphadenopathy. It differs from a primary chancre in that it is an ulcer rather than a granuloma and therefore has a soft feeling when gripped between the fingers. In addition, it is rarely single, it is invariably painful and tender, and the enlarged glands are tender at first and often break down later.

Lymphopathia venereum (lymphogranuloma or climatic bubo) the patient is febrile but not very ill, and the enlarged inguinal glands are tender. The surrounding tissues become inflamed.

(periadenitis) causing matting together of the affected glands to form a lobulated firm tender mass. Later in a proportion of cases multiple discharging fistulae develop.

In **bubonic plague** the patient is febrile and ill and the enlarged inguinal glands are firm and extremely tender. The skin over the glands becomes red and indurated prior to suppuration and sometimes glands in other regions such as axillae and neck are involved.

In **tick typhus** the thigh is a common site for the development of an eschar and in such cases the inguinal glands on that side are enlarged and tender. These signs together with fever, severe headache and maculo papular rash clinch the diagnosis.

In **Bancroftian filariasis** the femoral and inguinal glands become enlarged as a result of recurrent attacks of cellulitis affecting the oedematous leg or legs. A varicose condition of these glands may develop later in the course of the disease and is due to obstruction of the intra abdominal lymphatics. This is often associated with scrotal elephantiasis and chyluria.

One inguinal or femoral gland may become rapidly swollen and tender in lepromatous leprosy during an erythema nodosum reaction (see p 131).

Inguinal Ulceration A chronic ulcerative condition in one or both groins may follow gland suppuration secondary to soft sore and this is known as *ulcus molle serpigiosum*. The ulcer is slow spreading, has a fleshy base and its edges are ragged and deeply undermined. It should not be confused with lymphopathia venereum (climatic bubo) which gives rise to multiple small fistulous openings rather than to a large ulcer nor should it cause confusion with granuloma venereum (ulcerating granuloma of the pudenda) which does not involve the inguinal glands and is a chronic spreading granuloma, not a spreading ulcer. The granuloma which is slightly raised above the level of the surrounding skin has a red granular surface and shows a tendency to heal by scar formation in the centre. It slowly spreads peripherally to involve the external genitalia and the perineum. Often the external genitalia are involved first and the groin becomes affected by extension of the granulomatous process. Secondary ulcerations on the face and ears have been

described but the general health of the patient appears to be undisturbed

Rarely the groin may be the site of discharging sinuses in Bancroftian filariasis and a psoas abscess may rupture into one groin to form extensive ulcerations

Papular Conditions Moist flat papules present in the region of anus or vulva occur in congenital syphilis and in secondary syphilis and are sometimes found in other situations where the skin tends to be moist. Similar lesions occur in endemic syphilis. These are termed condylomata syphilitica and should not be confused with condylomata acuminata which are ordinary warts which happen to be situated in the genital or anal regions. These are often called venereal warts in spite of their non venereal nature. Care should be taken not to confuse external haemorrhoids with condylomata

Small shotty pink or violet papules with a red areola may occur on the external genitalia and perineum as rare ectopic lesions in schistosomiasis. They are separate or grouped, tend to appear in crops, may irritate at first and often develop a white spot at the centre which may become pustular and ulcerate

Scrotal Changes Elephantiasis of the scrotum occurs in conditions where the lymphatics draining the scrotum are chronically obstructed and the commonest cause in the tropics is Bancroftian filariasis. Hydrocele (effusion into the tunica vaginalis) is another common manifestation of this type of filariasis and the presence of fluid can be confirmed by transillumination and by eliciting fluctuation. By transillumination is meant a test for translucency by placing the light of a torch up against the back of the distended scrotum while the examiner inspects the scrotum from the front. This is best seen in a darkened room or by peering down a rolled up sheet of paper to exclude the light. Thickening of the scrotal wall may vitiate the test. Hydrocele can be differentiated from hernia by the absence of expansile impulse on coughing and by the fact that the examiner's fingers can be made to meet around the spermatic cord at the upper end of the scrotum. In addition it is often possible to reduce a hernial swelling back into the abdomen by manipulating it when the patient is lying on his back.



The scrotum may be enlarged by **oedema** in congestive heart failure and in nephritis but the oedema is not confined to the scrotum and involves the penis and other dependent parts

A nodular condition of the **spermatic cord** is sometimes encountered in Bancroftian filariasis but also raises the possibility of tuberculosis

A varicose condition of the scrotal veins noticed when the patient stands up is known as a **varicocele**. It is usually confined to the left side of the scrotum and is of no clinical importance but the abdomen should be examined to exclude a renal neoplasm which might be causing renal vein obstruction. Scrotal erythema is a common complication of typhus fevers and may sometimes lead to gangrene of the scrotum and prepuce.

The skin of the scrotum may be thickened and elephantoid in advanced onchocerciasis (see p 70 114) and may be so wrinkled as to resemble the surface of a shelled walnut

Testes and Epididymes

Testicular Enlargement Testicular pain and swelling of acute onset may occur in mumps gonorrhoea Bancroftian filariasis and during an erythema nodosum reaction in lepromatous leprosy It may also be caused by trauma or torsion and is a rare complication in severe malignant tertian malaria and in brucellosis

Chronic epididymo orchitis is either tuberculous or syphilitic Tuberculous infection causes the epididymis to become painlessly enlarged and to be irregular and craggy on palpation The vas deferens feels thickened and irregular (beaded) and the testis becomes involved later in the disease Syphilitic infection causes painless diffuse gummatous infiltration of the testis the epididymis usually being spared and the testis feels large heavy smooth and devoid of sensation The vas deferens feels normal

Malignant disease of the testis takes the form of a seminoma a gradually increasing painless swelling associated with an effusion often bloodstained in the tunica vaginalis and with thickening of the vas deferens The testis is enlarged heavy and devoid of sensation If untreated the growth invades the tunica and later invades the scrotal skin

When testicular enlargement of slow evolution and obscure aetiology occurs it would be wise to consider the possibility of schistosomiasis and to arrange for suitable diagnostic tests to be carried out (see p 151)

Testicular Atrophy This is a normal accompaniment of an undescended testis Atrophy occurring when the testis is in its normal position in the scrotum is usually associated with one of the following infections – mumps gonorrhoea tuberculosis leprosy or syphilis Rare causes include injury torsion compression of spermatic cord or of testis in filarial elephantiasis of the scrotum and testicular agenesis (Turner's syndrome and

the Klinefelter Albright syndrome) Turner's syndrome has been described on p 41 and in Klinefelter Albright syndrome the patient is of normal stature and is potent but he is infertile due to the fact that his semen is devoid of spermatozoa (azoospermia)

Examination of the Anus The anus should be inspected with the patient in the left lateral position. Fistulae in the perianal region call for investigations to exclude tuberculosis. External piles (haemorrhoids) appear as bluish prominent veins at the anal orifice and are of no importance. If one of these veins becomes thrombosed it produces a firm purplish tender swelling the size of a small grape. Internal piles may be permanently prolapsed or may appear when the patient is asked to strain down. A fissure can be seen when the anal margin is stretched and is exquisitely tender. The examiner's finger should be inserted gently to its full length after adequate lubrication and any palpable abnormality should be noted. A palpable mass attached to the rectal wall calls for further investigation to exclude carcinoma and a palpable narrowing of the lower end of the rectum raises a suspicion of lymphopathia venereum (climatic bubo).

When a newborn infant is being examined the practitioner should inspect the anal region to exclude anal atresia.

HANDS



Clubbing with
dilatation of
the finger
toes

Bulbous thickening of the soft tissues of the terminal phalanges with exaggeration of the natural curves of the nails is known as clubbing. Except in the rare familial form the toes are involved as well as the fingers but clubbed toes are not so easy to recognise because of the wide variation in the shape of normal toes.

Certain diseases of the lungs are commonly associated with clubbing and these include bronchiectasis abscess chronic empyema fibroid tuberculosis with cavitation pneumoconiosis actinomycosis cystic disease and carcinoma. Clubbing may also occur in paragonimiasis.

There are two cardiac conditions which commonly cause clubbing namely congenital cyanotic heart disease (morbus caeruleus) and subacute bacterial endocarditis. It occurs less commonly in congestive cardiac failure.

Intestinal causes include ulcerative colitis regional enteritis intestinal tuberculosis the sprue syndrome and multiple polyposis of the colon. Clubbing may occur in Hodgkin's disease when there is glandular enlargement within the abdomen or chest.

Well marked clubbing of the fingers in the absence of any similar changes in the toes is familial in origin. Clubbing of the fingers of one hand the other hand and the toes being normal is an important sign of an aneurysm of the aorta or of one subclavian or innominate artery.

When clubbing is due to intra thoracic disease it is sometimes part of a wider syndrome known as hypertrophic pulmonary osteo arthropathy. In this syndrome there occurs besides clubbing chronic periostitis of the long bones of forearms and legs. This may not give rise to any obvious symptoms or signs (apart from typical radiological changes) or may be associated with pain stiffness effusion tenderness along the shafts of the affected bones or elephantiasis of one or more limbs.

Dactylitis Dactylitis gives rise to a fusiform swelling of the proximal inter phalangeal joint giving the finger a sausage shape. It is an early development in rheumatoid arthritis affecting particularly the second and third fingers of both hands and this must be differentiated from a similar type of digital swelling which may occur in the course of four chronic infections namely leprosy tuberculosis syphilis and yaws. Dactylitis in leprosy occurs in the lepromatous type usually affects one or more fingers in both hands and other stigmata of the disease are always present (see p 128). Dactylitis in tuberculosis and in congenital syphilis usually affects only one finger whereas it is usually multiple in yaws. Trauma gout or sub periosteal haemorrhage in scurvy may cause a painful fusiform swelling of one inter phalangeal joint and must be considered in the differential diagnosis.

Painless sausage shaped fingers may occur in syringomyelia often with cyanosis and the fingers feel soft and fleshy when gripped – the succulent hand of Charcot. The diagnosis is confirmed by the complete absence of sensitivity to pain and temperature in the hands the scars of previous injuries wasting of the intrinsic muscles and by absence of any skin lesions or thickened nerves of leprosy.

Unlike rheumatoid arthritis which affects the proximal inter phalangeal joints of the fingers and later leads to intrinsic muscle wasting and ulnar deviation of the fingers osteo arthritis affects the distal joints. Small rounded bony swell



Am. J. Med.

ings which are often tender at first arise over the affected finger joints these are known as Heberden's nodes

In chronic gouty arthritis all the finger joints may become swollen and deformed and similar changes will be found in the metatarso phalangeal joints of the big toes If tophi are not present over some of the affected joints a search should be made for them on the ears or over the patellae and elbows

Gonococcal arthritis may affect several joints in the hands but more commonly affects sternoclavicular temporomandibular vertebral or tarsal joints. Sometimes a single large joint is affected. The sudden onset of joint pain and swelling is helpful in diagnosis particularly if there has been a history of gonorrhoea in the past.

Size and Shape of Hands Massive hands with enlarged knuckles and thickened skin may be a pointer to acromegaly and confirmatory evidence should be sought in the form of enlargement of the feet face nose tongue and lower jaw wide spacing of the lower teeth kypho scoliosis and bi temporal hemianopia.

One hand may be larger than the other in a condition known as hemihypertrophy in which one half (one side) of the body becomes enlarged.

Long slender fingers and toes occurring in a tall, thin individual are manifestations of a congenital defect known as arachnodactyly and often there are other congenital defects as well. A well known triad consists of arachnodactyly atrial septal defect and bilateral dislocation of the lens. In sickle cell anaemia the patient who survives to adult age is sometimes tall and thin with long fingers and toes.

Short fingers and a broad hand are found in mongolism cretinism and in achondroplasia.

Colour of Hands Melanosis and cyanosis of the skin have already been discussed (see p. 13) but it could be mentioned here that melanotic pigmentation is often most marked on the knuckles as in Addison's disease and mild degrees of cyanosis can be seen in the nail beds. Slight cyanosis and puffiness of the hands and feet is often a feature of lepromatous leprosy and is probably due to damage to sympathetic nerves in the skin. Moist cold hands often slightly cyanosed in a patient free from intra thoracic disease should warn the observant practitioner that the patient's troubles may have a psychiatric background. Hands which are subject to intermittent attacks of pallor or cyanosis induced by emotion or exposure to cold and followed by a hyperaemic stage are characteristic of Raynaud's disease. In severe cases the fingers later become thin.

tapering and stiff while the skin over them becomes smooth shiny and tightly stretched. This is known as sclerodactyly and a similar condition of the hands unassociated with previous attacks of vasospasm and associated with atrophy of the skin of arms, face, neck and scalp is diagnostic of scleroderma. In this disease the face becomes mask like and difficulty in swallowing is encountered.

Redness of the palms and fingertips the so called liver palms are often present in cirrhosis of the liver and call for an examination of the skin of the trunk for spider naevi (see p. 49). Remember that the liver though enlarged at first may become shrunken in the later stages of cirrhosis giving rise to a decrease in the normal area of liver dullness on percussion. Redness of the palms and soles may accompany feverish conditions but is particularly noticeable in dengue fever.

Hyperkeratosis Hyperkeratosis of the palms (or soles) is a characteristic feature of certain spirochaetal diseases – yaws, pinta and endemic syphilis (including bejel and njoвера). These three diseases share many similar clinical features and considerable help in differential diagnosis is obtained by a knowledge of the part of the world in which the infection was acquired. Yaws occurs in many parts of the tropics but pinta is mainly confined to Central and South America and endemic syphilis is found in Yugoslavia, Turkey, Syria, Iraq, Arabia, North Africa and South Central Africa. Yaws and endemic syphilis have the following manifestations in common: chronic ulceration of skin, mouth and nose, patchy depigmentation of skin, sabre tibiae and juxta articular nodes. In yaws there is an earlier stage in which typical framboesiform lesions occur on the skin in successive crops: these are rounded papules about 1–2 cm. in diameter and covered with a yellow crust. Deprived of its crust the little swelling is seen to be red in colour and like a raspberry – hence the name framboesia. Pinta is characterised by irregular circinate areas of dyschromic skin usually distributed symmetrically on hands, fore arms, legs and feet. Their colour may be black, brown, purple or slate and there may be fine desquamation. The final lesions are depigmented and without scales.

Hyperkeratosis of palms and soles is also a feature of chronic arsenic poisoning and in patients with light skins there is a brown pigmentation which is often dappled with small lighter areas the so called rain drop pigmentation.

A patch of keratosis may appear on the palm as a result of occupational trauma. Small patches of pigmented dry scaly thickened skin may develop on the face and hands of Europeans who spend much time out of doors in the tropics. These are known as solar or actinic keratoses.



Manengile n'posy

th g n f th ght
f om t be c lo d i p o
of 4th a d 5th f g s

Fascial Changes in the Hand One or both palms may be the seat of Dupuytren's contractures. These are thickened bands of fascia which stand out and cause the fourth and fifth fingers to be drawn towards the palms. Pseudo Dupuytren's contractures occur in yaws.

Congenital contractures usually affect both little fingers but may affect the ring fingers as well. In this familial condition the contracted fingers are involuntarily hyperextended at the metacarpo-phalangeal joints and are flexed at the first inter-phalangeal joints.

Wasting of Hand Muscles In claw hand there is wasting of the interossei the outer two lumbricals and the hypothenar muscles and all these intrinsic muscles are supplied by the ulnar nerve. The 4th and 5th fingers are particularly affected and these become involuntarily hyperextended at the metacarpophalangeal joints and flexed at the phalangeal joints. The most important cause of claw hand in the tropics is leprosy and the damaged ulnar nerve can be felt as a thickened cord which may be smooth or irregular just above the elbow. The possibility of trauma should not be overlooked either to the ulnar nerve in the arm or to the medial cord of the brachial plexus (C8 T1). This cord may be compressed by a cervical rib or may be torn when the arm is forcibly elevated above the shoulder – Klumpke's paralysis.

Claw hand may be due to pressure on the anterior nerve root of the first thoracic nerve (T1) within the spine the cause being arthritis disc degeneration fractured vertebra syphilitic pachymeningitis or a neurofibroma. Finally damage to anterior horn cells in the spinal cord must be considered possible causes being poliomyelitis syringomyelia progressive muscular atrophy amyotrophic lateral sclerosis cord tumour meningomyelitis and transverse myelitis.

When the intrinsic muscles supplied by the median nerve are paralysed in addition to those supplied by the ulnar nerve there is paralysis of the thumb and wasting of the thenar muscles as well as claw hand and wasting of the hypothenar muscles. This gives rise to *main en griffe* or ape hand and is commonly seen in leprosy.

Spasm of Hand Muscles Tetany is characterised by intermittent muscle spasm in which the hands and feet are most commonly involved. The hands assume a peculiar position known as *accoucheur hand* in which the thumb is strongly adducted across the palm and the extended fingers are firmly adducted towards the middle finger to form a cone. The spasm generally affects the arms also the wrists and elbows being flexed. At the same time the feet become plantar flexed and the knees extended and this combination of events is known as *carpopedal spasm*. The spasm may last only a few minutes or several hours. Two signs can be elicited in the intervals between spasms. Chvostek's sign is positive if the facial muscles twitch when the facial nerve is gently percussed in front of

the ear and Trousseau's sign is elicited if the hand assumes the typical accoucheur position when pressure is applied to the upper arm. Spasm of the glottis is another manifestation of tetany and occurs in children; it is termed laryngismus stridulus. Tetany occurs when the serum calcium becomes low and also in alkalaemia (alkalosis). Causes of hypocalcaemia include rickets, the sprue syndrome and hypoparathyroidism. Alkalaemia results from excessive ingestion of alkali, from repeated vomiting in pyloric stenosis and from hyper-ventilation.



THE NAILS

A transverse furrow on one or more nails may reflect a severe illness at the time the affected portions were being formed in the matrix. After a course of emetine treatment the patient's fingers may show a broad white transverse band across the nails together with redness of the skin at the base of the nails and fine longitudinal fissuring of the nails themselves. These are known as emetine nails and the white bands reach a point half way up the nails in about 3-4 months.

The nails take on a blue colour in chronic Atebrin (mepacrine) poisoning - see p 11.

In severe iron deficiency anaemia the nails become thin and brittle, lose their convexity and become hollow. This is known as koilonychia or spoon nails. Brittleness and splitting of the nails sometimes occurs in women who are in normal health, in some the cause lies in the frequent use of nail varnish, but in the majority the cause remains unknown. The possibility of calcium deficiency and of mild iron deficiency anaemia should be considered in these patients. Similar changes can occur in lichen planus.

Ringworm causes one or more nails to become opaque discoloured and brittle (tinea unguium) sometimes whitish material collects between the free edges of the nails and the skin and the distal portions break away unevenly

Psoriasis causes the nails to become pitted thickened and opaque whitish material accumulates under the free edge of the affected nails as in ringworm. An important point in differentiation between psoriasis and ringworm of the nails is that in the former condition the nails are affected on both hands and on both feet symmetrically

Eczema of one or more fingers may cause irregular grooves and discolourations on the nails of the affected fingers

In secondary syphilis two or more nails on each hand may become thinned flattened brittle irregular and opaque

Where fingers have become tapering as a result of trophic changes due to Raynaud's disease sclerodactyly or leprosy the nails become narrowed curved and beak like

Petechiae in the nail bed are an important sign in subacute bacterial endocarditis and look like small splinters when the nail is gently pressed against the nail bed. Such splinter haemorrhages can however be seen in any conditions associated with purpura the most common tropical cause being scurvy. They commonly occur in acute trichiniasis

Inflammation of the fold of skin around the nail is known as paronychia and tends to affect those whose hands are continually being immersed in water. A common agent in such infections is a fungus *Candida albicans* and sometimes the affected nails become opaque discoloured and irregular – moniliasis of the nails

FEET

Sodden white skin in the clefts between the toes is indicative of the quiescent phase of *tinea pedis* and this may alternate with active phases when there is irritation or pain in the ball of the foot or between the toes accompanied by redness and vesiculation

A peculiar band like groove may slowly develop around the base of the little toe in dark skinned races almost exclusively in adult males leading to bulbous enlargement of the distal portion of the toe with loss of the nail and gradual destruction of the phalanges of the affected toe. This condition which is known as *ainhum* is painless is usually bilateral and has an unknown aetiology. The fleshy excrescence at the end of the affected toe becomes increasingly mobile and eventually drops off. Finger involvement is rare.

The female chigger flea (*Tunga penetrans*) when gravid penetrates the skin of the foot and becomes anchored in the subcutaneous tissue. The abdomen of the flea enlarges to form a small irritating pea like elevation of the skin at the site of penetration out of which the flea will eventually discharge her ova.



M d fo t (my t m)
(p f J E M ck M i d)

A h m



A localised hyperkeratosis occurs under one metatarso phalangeal joint usually the first or the fifth in diseases causing loss of pain sensation in the feet, such as **leprosy** **tabes** and **diabetes**. This sooner or later becomes softened and broken down to form a **trophic ulcer**. Once formed a trophic ulcer tends to enlarge and deepen owing to repeated painless trauma but can always be healed by a period of complete rest from weight bearing.

The metatarso phalangeal joint of one big toe is usually the site of attacks of **acute gouty arthritis** in the early stages of the disease the affected joint being swollen and extremely tender, and the surrounding skin being red and shiny.

Mycetoma (Madura foot) is characterised by a slow growing painless granulomatous tumour on one foot which develops sinuses from which the typical coloured grains are discharged. It is from the colour of these grains that the names **black**

Mo sy foot



yellow and red mycetoma are derived. As the infection spreads into the substance of the foot the bones become infiltrated and destroyed so that the whole foot becomes greatly enlarged and distorted. It remains a local infection and there is no systemic disturbance.

Massy foot is a localised mycotic infection in which the foot and ankle become covered with dense warty looking outgrowths but the sole is spared. A somewhat similar condition can occur in elephantiasis but is not due to a fungal infection. The excrescences are spiky rather than warty and can be scraped away with a blunt instrument. This is known as lymphostatic verrucosis.

In the secondary stage of yaws lesions on the soles cause erosion and fissuring. Walking is extremely painful and the patient's laboured crab like gait has given rise to the name crab yaws.

Bilateral oedema of feet and ankles calls for exclusion of renal or cardiac disease. Anaemia should be considered especially hookworm anaemia in the tropics. Other causes include beriberi, protein deficiency (including starvation and kwashiorkor), thyroid disease, liver cirrhosis and various allergic states. Epidemic dropsy can simulate beriberi but peripheral neuritis is absent. It is caused by mustard oil be-



coming contaminated with the poisonous seeds of *Argemone mexicana* and has occurred in epidemic in India and in other tropical countries in which Indians are settled. Oedema of the ankles probably due to vaso motor disturbance is often a presenting symptom in lepromatous leprosy. The abdomen should be examined to exclude pregnancy or other abdominal mass. Slight oedema of feet or ankles may occur after a long air journey this is due to the fact that the legs have been kept immobile in a sitting position for a long time thus impeding the venous return from the lower limbs. Heat oedema of feet and legs is common in Europeans on their first visit to the tropics.

In unilateral oedema the leg should be examined for varicose veins and should be carefully palpated for tenderness suggestive of thrombophlebitis. Oedema of one foot or ankle may be due to the presence of a Calabar swelling due to infection with the filarial worm *Loa loa* (see p 19).

Lymphoedema is discussed on p 113.



T. T. T. T. T.



Hygrom (bu so) of knee



Jit -
atclir d

LEGS

Ulceration A **tropical ulcer** is a single large oval shaped ulcer usually situated on the lower part of the leg in the ankle region or on the dorsum of the foot. It has a raised edge and a grey sloughing base pain is often present but constitutional symptoms are absent.

A **veld sore** is a punched out circular small painful ulcer with an undermined edge which is thickened and cyanotic. It is single or multiple and is situated on exposed skin such as face arm leg or back of hand. If contaminated with diphtheria bacilli the sores may be complicated by paralysis or paresis of the palate ocular muscles or limbs. In some instances a direct relationship can be observed between the site of the lesion or lesions and the site of the palsy.

An **oriental sore** (cutaneous leishmaniasis) commences as a nodule which slowly enlarges over several months before becoming an ulcer. It is more often single than multiple and although often situated on the leg may occur on any exposed area such as the face arm and back of hand or foot. The ulcer is roughly circular or oval is usually not more than 3 cm

in diameter and is painless. It is characteristically an ulceration which is taking place in a granuloma and is therefore slightly raised above the general level of the skin, more so at the edges. It is covered with a dried crust which, being removed, reveals a sharp edge and an irregular base covered with reddish yellow granulations.

An ulcer on the leg due to yaws may be the primary sore or mother yaw – a painless raised granuloma about 3 cm or more in diameter with central necrosis and crusting. It may however be a lesion of tertiary yaws in which case there are likely to be more than one ulcerating gumma. Search should be made for other stigmata of tertiary yaws such as hyperkeratosis of palms or soles, juxta articular nodes (rounded subcutaneous nodules occurring at the knees, hips or elbows), gangosa (ulceration of the nose and nasopharynx), sabre tibiae and skin depigmentation. It should be remembered that gummatous ulcers may occur in the later stages of syphilis whether venereal or endemic.

G e a w m



G w m





Elephantiasis of left leg

hausen's disease) and is called elephantiasis neuromatosa it arises as a result of overgrowth of skin over a plexiform neuroma and can affect the temple one upper eyelid the back of the neck or a limb Another rare form of elephantiasis occurs in hypertrophic pulmonary osteoarthropathy (see p 96)

In advanced onchocerciasis the skin of the legs can become greatly thickened and wrinkled resembling crocodile skin The skin of the scrotum may be similarly thickened or hanging groins may be present skin sacs in each groin containing enlarged lymph glands secondary to septic dermatitis Onchocercal nodules should be searched for in the pelvic region In addition examination of other areas particularly the back will reveal the Morocco leather appearance of the skin typical of advanced onchocerciasis The eyes should be examined for evidence of keratitis (see p 23)

Tenderness in the Legs

An ill febrile patient whose calf muscles are tender even on light handling requires investigation for leptospirosis – of which Weil's disease is the commonest form Look for red conjunctivae herpes labialis jaundice and tender liver enlargement

A non febrile patient whose calves are tender on being gripped between finger and thumb is suffering from **peripheral neuritis**. The knee and ankle jerks will be found to be absent vibration sense will be diminished and there may be muscle wasting and sensory impairment over the legs. If untreated foot drop is likely to develop. **Beriberi** is the commonest cause of peripheral neuritis in the tropics and the heart should be examined for confirmatory signs (see p 58)

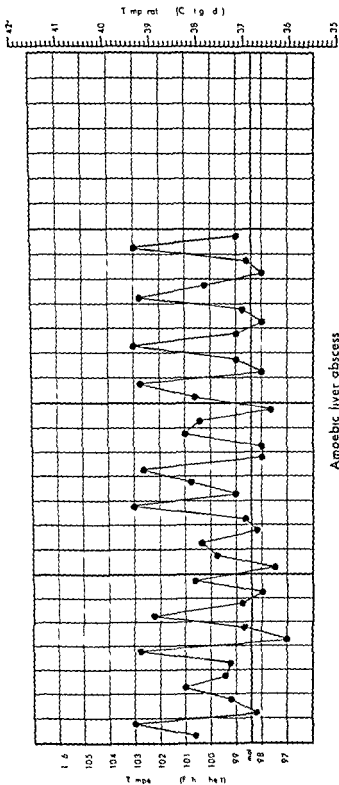
Tenderness over the periosteum of the tibiae is a sign of African **trypanosomiasis** known as Kerandel's sign and it can be elicited over the ulnar periosteum as well. It is a peculiar form of hyperalgesia in which the slightest blow causes severe pain after a short interval it might well be called delayed hyperalgesia. Early signs of trypanosomiasis should be looked for (see p 135)

BODY TEMPERATURE

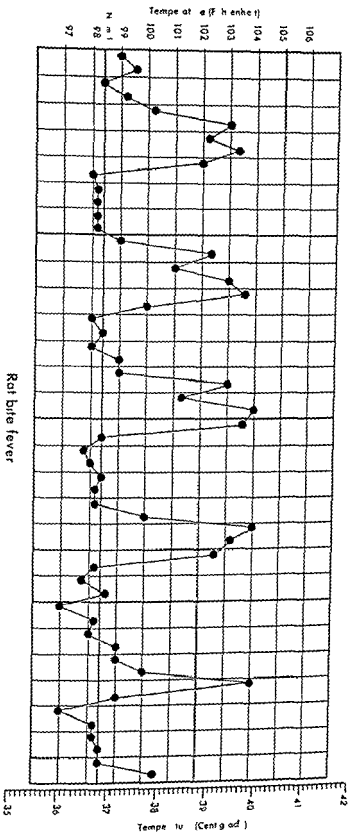
Before considering a raised body temperature to be pathological the practitioner should exclude a physiological cause. This is particularly important in the tropics where temperatures of healthy persons may be raised by as much as 1°F (0.55°C) in the hottest months of the year oral temperatures lying between 98° and 100°F (36.7° and 37.8°C) instead of the usual 97° – 99°F (36.1° – 37.2°C). Other physiological causes of raised body temperatures are exercise eating a large meal, and emotional disturbance particularly in children. Women may have an increase in temperature of 0.5° – 1°F (0.28° – 0.55°C) during part of the menstrual cycle commencing one week before menstruation commences. The oral temperature may be temporarily raised after a hot drink.

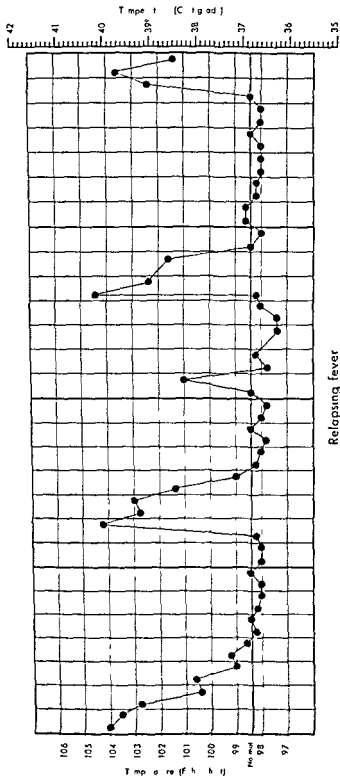
In general rectal temperatures are more accurate than oral recordings in registering body temperature and are 0.5 – 1°F (0.28 – 0.55°C) higher. This is not always so however for experiments on healthy persons in the tropics have shown that there are occasions when rectal recordings can be as much as 3°F (1.7°C) higher than oral readings or may even be 1°F (0.55°C) lower.

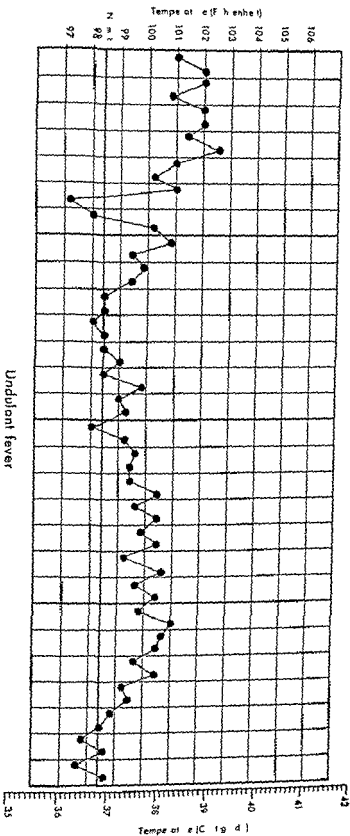
If a patient is suspected of having heat stroke it is essential to record the rectal temperature and to have frequent readings while carrying out treatment to lower the body temperature. Similarly temperatures should be recorded rectally in cholera and in algid malaria.



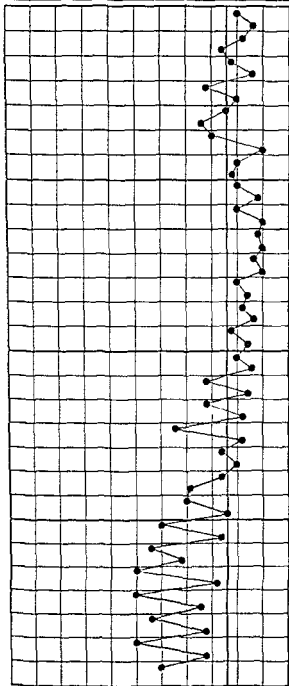
Amoebic liver abscess





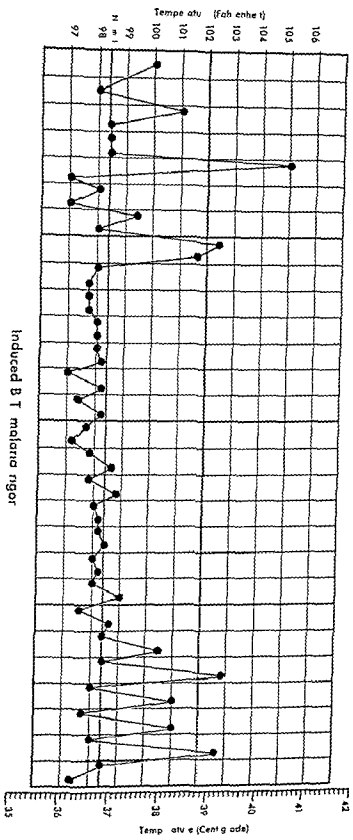


Temperature (C) (F)

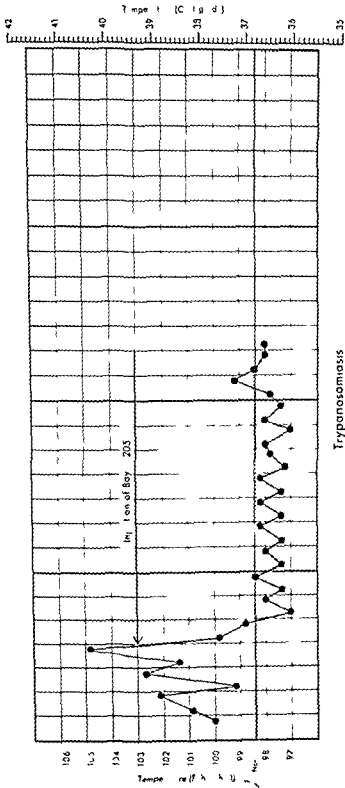


Kala azar

Induced B T malaria ngor

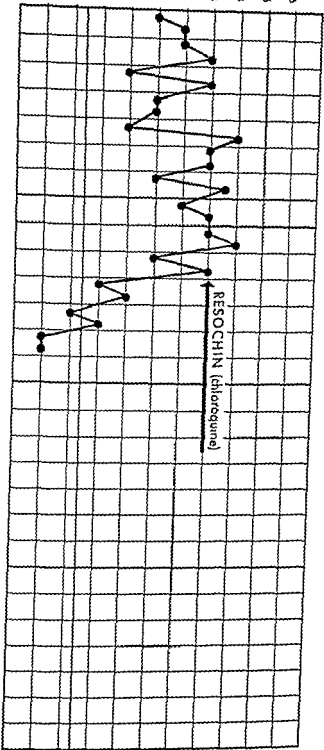


Trypanosomiasis



Temperatu (C 1 g ad)

Malignant tertian malaria





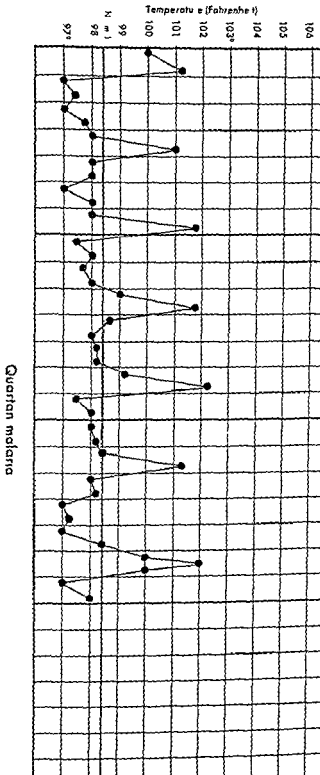
DERMATOSES IN THE TROPICS

15

rash which commences a few hours
n fresh water
cies of sch
or
The
Some of these
hers can cause
only the
de

3

Temp r e (C e l s i u s)



Quartan malaria



Small papules on the neck



Small papules on the face

APPENDIX-1

CERTAIN EXANTHEMATA AND DERMATOSES COMMONLY ENCOUNTERED IN THE TROPICS

CERCARIAL DERMATITIS

This is an irritating popular rash which commences a few hours after bathing or wading in fresh water contaminated with the cercariae of various species of schistosomes. Some of these schistosomes may inhabit birds or animals others can cause disease in man (schistosomiasis). The rash affects only those areas of skin which have been in contact with water and reaches its maximum intensity in 2 or 3 days to fade in about a week.

DENGUE

The rash usually appears about the 4th day of the illness soon after the patient has enjoyed a short remission of his fever. It commences on the hands sometimes on the feet as well and rapidly spreads to the limbs and trunk but usually not to the face. The rash appears at first as isolated circular erythematous macules followed by coalescence to produce large areas of erythema with patches of pale (unaffected) skin in between. This appearance has been described as half way between measles and scarlet fever. The rash fades on pressure. It lasts about 2-3 days and fades in the order of its appearance. Palms and soles are not involved but may show a carmine flush. The face is flushed and the conjunctivae are injected.

LARVA MIGRANS (CREEPING ERUPTION)

The larvae of *Ancylostoma caninum* and *A. braziliense* (the hookworms of dogs and cats) can penetrate human skin which has come into contact with infected soil or sand. A red itchy papule develops at the point of entry and within 2 or 3 days a red wavy slightly elevated line begins to creep across the skin at the rate of about 2 cm every 24 hours. This marks the track of the larva burrowing in the skin and is intensely irritating. Scratching often leads to secondary infection. The affection usually lasts for several months.

The larvae of botflies of the genus *Gastrophilus* can cause a similar type of creeping eruption.

Another form of larva migrans occurs as a complication of an intestinal infection with *Strongyloides stercoralis* and usually attacks the buttocks and trunk.

LEPROSY (HANSEN'S INFECTION)

a Tuberculoid leprosy The lesions take the form of macules or infiltrated (raised) lesions which are few in number and are asymmetrically situated on the body. Macules are erythematous on light skins hypopigmented (not depigmented) on dark skins. have a dry surface have edges which are clearly defined and show loss of touch and temperature sensation.



l p male sl poy



l p m l p y



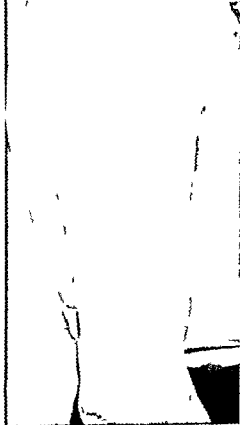
l p amar l p ro y



l p rom l p y



T b e l d l p o s y



8 d l a (d m o p h u s) l p o s y



L p m t 3 e p o s y
f l e o



L p m t o l p o y

(except on the face) Infiltrated lesions are erythematous with sometimes a coppery or purple hue have a dry and rough surface which may be irregular or pebbled are sometimes scaly have well marked sensory loss and have edges which are raised and clear cut while the centres tend to be flattened. Peripheral extension may give rise to a large annular lesion. One or more peripheral nerves can usually be palpated as thickened cords in the region of the lesions. Lesions of tuberculoid leprosy may undergo reactional hyperaemia and swelling giving the appearance of erysipelas but fever and constitutional symptoms are absent. During such a reactional phase there may be pain and swelling in one or more of the affected nerves.

b Lepromatous leprosy The lesions take the form of macules, infiltrations, papules and nodules, all of which may be present in the same patient by the time the disease has become advanced. The earliest lesions are macules and these tend to be ignored by the patient as they are difficult to see and are not anaesthetic. They are small and multiple, have a smooth and shiny surface and their edges are indistinct. They are erythematous in light skins and are erythematous or faintly hypopigmented in dark skins.

Infiltrated lesions are erythematous with sometimes a coppery or purple hue, are raised in the centre and slope away peripherally to merge imperceptibly with the surrounding skin, have a smooth and shiny surface and do not exhibit sensory loss (unless situated in a region of skin which is anaesthetic from peripheral nerve damage). Papules and nodules may be of normal skin colour or may be brownish or erythematous; they have a predilection for the face and buttocks. Note that the skin lesions of lepromatous leprosy differ from those of tuberculoid in that they are multiple, are symmetrically distributed and are not anaesthetic.

An allergic phenomenon known as erythema nodosum leprosum may occur in lepromatous leprosy in which crops of transient bright pink nodes and raised patches appear on the skin, most commonly on arms and legs. They appear suddenly, usually in the evening, the smaller ones disappearing by the following morning and the larger ones taking days or weeks to fade and leave a blue stain in the skin. The patient often complains of burning discomfort in these lesions and pres

sure on them with the finger may be painful. Their bright pink colour disappears immediately slight pressure is exerted on them but it returns as soon as the pressure is released. When numerous they are accompanied by constitutional symptoms which include intermittent fever, severe nerve pains, arthralgia, bone pains, acute iridocyclitis, acute epididymo orchitis, rhinitis, epistaxis, lymphadenitis, insomnia and mental depression.

c. Borderline (dimorphaus) leprosy This is the type of leprosy occurring in patients who fall in an intermediate category not possessing the good defence mechanism of those with tuberculoid leprosy nor the complete lack of immunity which characterises lepromatous leprosy. Skin lesions are macular or infiltrated, the earliest lesions being macules which are faintly erythematous in light skins and are hypopigmented in dark skins. They have a predilection for the back or the thighs and careful testing will reveal loss or impairment of sensation in some if not in all. Infiltrated lesions are moderate in number, anaesthetic, asymmetrical in distribution, their erythema has an admixture of purple or brown, their surface is smooth and often shiny and they slope away peripherally from raised centres. Their edges are well defined in plates and indefinite in others. Some of the infiltrations may take the form of bands, annular lesions and small nodules.

As in tuberculoid leprosy infiltrated lesions become erysipeloid during reactional phases followed by desquamation.

MEASLES (MORBILLI)

After 3 or 4 days of coryza, cough and conjunctival injection small dark red macules appear behind the ears and at the junction of the forehead and scalp, shortly to be followed by maculo papules on the face, neck, upper arms and chest, later spreading to the back, abdomen, forearms, hands, thighs and lastly to the feet. The maculo papules fuse to form blotches with areas of clear skin between, the rash being most profuse on the face and trunk. In severe measles the skin may be entirely covered and the rash may become petechial. The face becomes swollen and the eyes more bloodshot. The rash becomes dull and coppery before it fades in the order of its appearance. Measles is not so easily recognized on a coloured skin.

MILIARIA RUBRA (PRICKLY HEAT)

This affects white persons in the tropics and favours the areas of skin which are most subject to sweating and which are rubbed by clothing or are in contact with opposing skin surfaces e.g. axillae mid abdomen and under breasts. The affected areas develop tiny vesicles and closely set red papules giving the impression of white sand sprinkled on to a bright red surface. The irritation and burning discomfort caused by the rash is very distressing particularly as it interferes with sleep.

ONCHOCERCIASIS

In the early stages of onchocerciasis an erythematous maculopapular skin rash which is intensely irritating develops on the skin. It may be localised to only one area such as the back or may affect more than one area. The commonest sites are the back, buttocks, thighs and upper arms and scratch marks are invariably present. These may cause secondary infection and lymphadenopathy. At this early stage there are not likely to be any visible nodules but small nodules of firm consistency and about the size of sunflower seeds may be palpated in the subcutaneous tissue around the iliac crests. The eyes should be examined with a torch and corneal loupe for small milky spots in the superficial portions of the corneae.

PELLAGRA

The rash usually occurs on areas of skin exposed to sunlight but may be provoked by pressure or friction. It is commonly found on the back of the neck (Casal's necklace) and on the backs of hands and wrists. It commences as an erythema with a distinct line of demarcation and later the affected areas become brown, rough and scaling. The rash may also affect the face extending across the bridge of the nose and on to the cheeks in a butterfly pattern. The bilateral symmetry of the lesions is striking.

SMALLPOX (VARIOLA)

During the first 2 days of the illness transient scarlatiniform or morbilliform rashes may appear especially on the buttocks and upper thighs. A petechial rash at this stage with bleeding into the skin and from the mucous membranes signifies a haemorrhagic type of smallpox which is invariably fatal.

On about the 3rd day of the illness pin head macules appear and last for a few hours before becoming dark pink papules 3 mm or more in diameter each one surrounded by a pale pink areola. Papules continue to appear for the next 2-3 days then rapidly become vesicular the conversion of papules to vesicles commencing with those lesions which were the earliest to appear. As the fluid tension in the vesicles decreases some of them become dimpled or umbilicated. After 2 days the vesicles become pustules which enlarge and may reach a diameter of 10 mm. The pustules last for about 4 days before drying up to form dark brown scabs.

To avoid mistakes in diagnosis it is important to pay particular attention to the distribution of the rash. The earliest skin lesions appear on the face to be followed by others on the arms and hands then on the trunk and finally on the legs and feet. The greatest number of lesions occur on the face hands forearms feet and legs while the more central regions like the trunk upper arms and thighs are relatively spared. The rash therefore has a centrifugal distribution. Also the rash favours the extensor rather than the flexor surfaces of the limbs and avoids concavities and protected parts such as axillae. Lesions are commonly present on the palate and fauces.

As smallpox is commonly misdiagnosed as chickenpox it is important to remember that the distribution of the rash in chickenpox is the exact opposite being centripetal and not centrifugal also the lesions in chickenpox appear in crops and an examination of any one region such as the face will reveal lesions in different stages of development i.e. papules vesicles and pustules.

The practitioner should appreciate that the attack of smallpox may be modified by a previous vaccination which has rendered the patient partially immune. The rash will be scantier but it will conform to the classical distribution which has just been

described. The individual lesions however are more superficial and some may fail to progress through the various stages from macule to pustule. That is to say some will regress after reaching the papular stage and others will not develop beyond the vesicular stage thus giving the appearance of several crops and possibly leading to a wrong diagnosis of chickenpox. It should be remembered that the patient with modified smallpox is far more dangerous to the community than one with a prostrating attack for he may not feel ill enough to keep to his bed and any non-immunes who come into contact with him (or with his clothes) are liable to develop virulent and fatal smallpox.

TRYPANOSOMIASIS AFRICAN

Erythematous macules frequently annular and with irregular margins may occur anywhere on the skin but are most common on the chest back and thighs. The rash comes and goes tends to be brought out by heat and is often unobserved by the patient. Although it occurs more commonly in the early stages of the infection it may sometimes be found in the later stages when there are signs of involvement of the central nervous system. In the early stage of the infection search should be made for a trypanosomal chancre a red nodule surrounded by oedema which may be present on one arm or other exposed area of skin.

TYPHOID AND PARATYPHOID

The rash in typhoid is sparse and consists of discrete rose coloured spots which are slightly raised and fade momentarily on pressure. They appear in successive crops each crop fading in a few days. Although usually present on the upper abdomen the spots may sometimes be found in addition on other sites such as the back.

The rash in paratyphoid is similar excepting for its tendency to be more profuse more widespread and to have a deeper colour. It may be confused with the rash of typhus and when the face is involved it is sometimes mistaken for measles. The rash in typhoid and paratyphoid first appears on about the 8th day of the illness.

SMALLPOX (VARIOLA)

During the first 2 days of the illness transient scarlatiniform or morbilliform rashes may appear especially on the buttocks and upper thighs. A petechial rash at this stage with bleeding into the skin and from the mucous membranes signifies a haemorrhagic type of smallpox which is invariably fatal.

On about the 3rd day of the illness pin head macules appear and last for a few hours before becoming dark pink papules 3 mm or more in diameter, each one surrounded by a pale pink areola. Papules continue to appear for the next 2-3 days then rapidly become vesicular the conversion of papules to vesicles commencing with those lesions which were the earliest to appear. As the fluid tension in the vesicles decreases some of them become dimpled or umbilicated. After 2 days the vesicles become pustules which enlarge and may reach a diameter of 10 mm. The pustules last for about 4 days before drying up to form dark brown scabs.

To avoid mistakes in diagnosis it is important to pay particular attention to the distribution of the rash. The earliest skin lesions appear on the face to be followed by others on the arms and hands then on the trunk and finally on the legs and feet. The greatest number of lesions occur on the face, hands, forearms, feet and legs while the more central regions like the trunk, upper arms and thighs are relatively spared. The rash therefore has a centrifugal distribution. Also the rash favours the extensor rather than the flexor surfaces of the limbs and avoids concavities and protected parts such as axillae. Lesions are commonly present on the palate and fauces.

As smallpox is commonly misdiagnosed as chickenpox it is important to remember that the distribution of the rash in chickenpox is the exact opposite being centripetal and not centrifugal also the lesions in chickenpox appear in crops and an examination of any one region such as the face will reveal lesions in different stages of development i.e. papules, vesicles and pustules.

The practitioner should appreciate that the attack of smallpox may be modified by a previous vaccination which has rendered the patient partially immune. The rash will be scantier but it will conform to the classical distribution which has just been

described. The individual lesions however are more superficial and some may fail to progress through the various stages from macule to pustule. That is to say some will regress after reaching the papular stage and others will not develop beyond the vesicular stage thus giving the appearance of several crops and possibly leading to a wrong diagnosis of chickenpox. It should be remembered that the patient with modified smallpox is far more dangerous to the community than one with a prostrating attack for he may not feel ill enough to keep to his bed and any non-immunes who come into contact with him (or with his clothes) are liable to develop virulent and fatal smallpox.

TRYPANOSOMIASIS AFRICAN

Erythematous macules frequently annular and with irregular margins may occur anywhere on the skin but are most common on the chest, back and thighs. The rash comes and goes, tends to be brought out by heat and is often unobserved by the patient. Although it occurs more commonly in the early stages of the infection it may sometimes be found in the later stages when there are signs of involvement of the central nervous system. In the early stage of the infection search should be made for a trypanosomal chancre, a red nodule surrounded by oedema which may be present on one arm or other exposed area of skin.

TYPHOID AND PARATYPHOID

The rash in typhoid is sparse and consists of discrete rose coloured spots which are slightly raised and fade momentarily on pressure. They appear in successive crops, each crop fading in a few days. Although usually present on the upper abdomen the spots may sometimes be found in addition on other sites such as the back.

The rash in paratyphoid is similar excepting for its tendency to be more profuse, more widespread and to have a deeper colour. It may be confused with the rash of typhus and when the face is involved it is sometimes mistaken for measles. The rash in typhoid and paratyphoid first appears on about the 8th day of the illness.



Rocky mountain
potted fever

TYPHUS

a Epidemic Typhus (Exanthematic Typhus) From the 3rd to the 5th day of the illness small dark pink macules varying in size and shape appear on the anterior folds of the axillae and on the loins. The rash spreads to the chest, back, shoulders and limbs, the greatest concentration of macules being on the loins, abdomen and back where they appear in clusters. The face is spared but has a dark red flush and later becomes swollen. The conjunctivae are injected. The macules do not appear in successive crops and the skin between them may show a dusky mottling called *subcuticular mottling*. In a day or two the macules become dull rusty red followed by colour change to slate or grey before disappearing but often this sequence is interrupted by the rash becoming dark red and petechial.

b Scrub Typhus (Tsutsugamushi) On the 4th or 5th day of the illness sometimes later the rash first appears. Commonly it is first noticed on the chest or abdomen and it spreads to the neck, face and limbs. It is most pronounced on the trunk. The

rash takes the form of small reddish macules which increase in size and number until widespread giving the skin a mottled appearance. Palms and sole are unaffected. When widespread the rash consists of large and small macules and papules or there may be a diffuse erythema with superadded macules and papules. The rash reaches its maximum intensity in a few days and gradually fades. Desquamation takes place at this stage and is best seen in dark skins. The skin should be searched for evidence of an eschar, particular attention being paid to axillae and groins. This is a dark scab about 2-4 mm in diameter with a red areola. The regional lymph glands are enlarged and tender.

c Tick Typhus The rash appears between the 5th and 7th day of the illness. It takes the form of rose pink macules superimposed on a diffuse erythema and is often first noticed on the front of the chest and abdomen. It spreads to the face then to the arms and legs (including palms and soles) but in Rocky Mountain spotted fever the rash usually first appears on the wrists and ankle. During the course of the next few days the rash becomes maculopapular with some of the macules becoming raised and changing colour to dark bluish red. In all forms of tick typhus except Rocky Mountain spotted fever an eschar is likely to be found at the site of the infecting tick bite (see scrub typhus).

APPENDIX 2

MACROSCOPICAL EXAMINATION OF THE URINE

Normal urine is transparent and the colour of amber or pale sherry. It is paler than normal after drinking a large quantity of water or when the kidneys are diseased and have lost their normal concentrating power. The colour is darker than normal when the urine is concentrated as for example in dehydration from any cause. Opalescence is normal if it can be removed by filtration; if it persists after filtration it is due to the presence of bacteria. Milky urine is indicative of chyluria.

After urine has stood for some time a deposit may collect at the bottom of the jar due to normal ingredients such as phosphates and urates. Urates give a pink colour to the deposit and this may be noted in dehydration after hard exercise, in fevers, in cirrhosis of the liver, in heart failure and in myeloid leukaemia.

An abnormal colour of urine may be due to exogenous or endogenous pigment.

Exogenous Pigment

Colour of Urine	Cause
Brown when acid Orange when alkaline	Rhubarb Senna
Pink with green fluorescence	Eosin
Red or pink	Beetroot
Cherry red	Amidopyrin
Green	Methylene blue (de Witts pills)
Green brown	Carbolic acid
Blue green fluorescence	Quinine
Green yellow if acid Pink if alkaline	Santonin
Yellow	Flavine or picric acid to wounds
Yellow	Miracil® D (lucanthone hydrochloride)
Yellow	Atebrin® (mecaprine)

Endogenous Pigment

Smoky May be a red tinge	}	Blood
Brown black on standing		
Black or brown black		Haemoglobinuria
Port wine colour or brown		Porphyria
Orange yellowish brown or yellowish green		Jaundice
Normal when passed Black on standing		Alkaptonuria
Brown black (due to melanin)		Malignant melanoma

APPENDIX 3

Naked Eye Examination of Faeces

Disease	Macroscopic	Odour	Organism
Pancreatitis	Bulky yellowish white liquid or semisolid should set in half an hour after passage without blood or mucus	Cheesy	Nil
Coeliac Disease & Idiopathic Steatorrhoea	Liquid or semisolid bulky yellowish white faeces without blood or mucus	Very offensive	Nil
Tropical Sprue	Bulky fermenting yellowish white liquid or soft solid faeces like rolls of farmyard butter without blood or mucus often contains bubbles	Sour penetrating like burned paint	Nil
Bacillary Dysentery	Thin blood stained mucus or blood & mucus with greenish liquid faeces Sometimes viscid & sticky like Red currant jelly	Not offensive Smells like laundered linen	Dysentery bacilli by cultural methods
Typhoid and Paratyphoid	Liquid brown pea soup faeces occasionally with a streak of blood & mucus	Very offensive like butcher's meat	Typhoid and paratyphoid bacilli by cultural methods
Cholera	Thin watery rice water stools without blood or mucus	Odourless	Cholera vibrio by cultural methods
Amoebic Dysentery	Thin layer of mucus streaked with blood intermingled with normal or liquid faeces looks like Anchovy sauce In chronic stage liquid faeces without blood or mucus	Offensive	<i>Entamoeba histolytica</i> vegetative active forms cysts in chronic stage

Disease	Macroscopic	Odour	Organism
Balanitidis Dysentery	Liquid brown faeces intermingled with blood & mucus – resembles amoebic dysentery	Very offensive	<i>Balanitidis coli</i> confirmed by staining methods
Giardiasis (Lambliasis)	Mostly mucus or liquid faeces intermingled with mucus – but may be pale & pasty resembling sprue	Inoffensive	<i>Giardia (Lambia)</i> confirmed by staining methods
Other Flagellate Infections	Gelatinous copious mostly liquid faeces	Offensive	<i>Trichomonas hominis</i> <i>Chilomastix mesnili</i> etc
Schistosomiasis	Semisolid chocolate coloured faeces thick blood & mucus often in clumps or intermingled with faeces	Offensive	Eggs of <i>Schistosoma haematobium</i> <i>S. mansoni</i> or <i>S. japonicum</i>
Mucous Colitis	Strings ofropy mucus intermingled with liquid or semi solid faeces	Offensive	Nil
Membranous Colitis	As above – sometimes shreds of membrane or more rarely complete casts of intestinal mucosa	Offensive	Nil
Spastic Colitis	Ribboned solid faeces	Normal odour	Nil
Ulcerative Colitis	Viscid mucus & abundant dark blood with or commonly without liquid faeces Sometimes abundant creamy mucus	Very offensive usually smells of decomposing blood	Nil

Disease	Macroscopic	Odour	Organism
Polyposis	Dark blood with small quantity of mucus No visible pus Usually numerous stools	Offensive	Nil
Polypus	White clean mucus resembling sputum with or without faeces	Not usually offensive	Nil
Intestinal Tuberculosis	Greenish brown liquid faeces usually bulky – sometimes intermingled with flecks of blood & mucus	Very offensive	Tubercle bacilli confirmed by appropriate staining
Food Poisoning	Greenish liquid faeces with undigested food particles	Very offensive	<i>Salmonella enteritidis</i> & other members of the group
Mushroom Poisoning	Very liquid almost choleraic faeces	Not offensive	Spores of fungus can be recognized

LABORATORY DIAGNOSIS

The laboratory is a good servant but a bad master

This section of the Appendix is designed to assist the practitioner in the tropics by advising him as to the tests which will help him in making a diagnosis. The diseases are arranged in alphabetical order for ease of reference.

General Notes

Blood culture It is not satisfactory to send blood to a distant laboratory for blood culture as the chances of contamination are too great. The practitioner should consult the pathologist at the central hospital regarding the culture media which he should keep in stock and he should then be able to inoculate the cultures himself by the bedside. The cultures can then be sent to the laboratory.

Animal inoculation When animal inoculation is necessary to establish a diagnosis it would be advisable to send the patient to the central hospital.

Complement fixation tests and agglutination reactions It is useless to send whole blood to a distant laboratory for these tests as it invariably becomes haemolysed in transit. The practitioner should collect about 10 ml of venous blood and should centrifuge it. The serum can then be collected and sent to the laboratory. Note that agglutination reactions do not become positive until the second week of illness and the examination should be repeated at intervals in order to observe a rising titre.

Blood smears The diagnosis of a number of tropical diseases depends upon the microscopical examination of stained blood films and the practitioner is advised to make one thick film and two thin films. A thick film is made by placing a drop of blood on the centre of a glass slide and holding the stabbing needle parallel with the surface of the slide the drop is spread out with a gentle circular motion into a circular film about 1.5 cm in diameter.

Two glass slides are required for making a thin film. A drop of blood is placed at one end of the first slide and the second slide is then moved across the first slide (making an angle of about 45 degrees with it) until it reaches the drop of blood this causes the blood to spread out into a straight line and the second slide is then pushed across the whole length of the first slide in a single smooth and rapid movement. The blood is pulled across the first slide by this method.

The practitioner should ensure that all glass slides are free from grease. The slides should be washed with hot water and soap and then should be dried with a smooth towel after rinsing in hot water.

Citrated or oxalated blood When trypanosomes or filarial embryos are too scanty to be found in blood films a sample of citrated or oxalated blood should be sent to the laboratory. 5 ml of venous blood is placed in a small bottle containing anticoagulant such as Wintrobe's oxalate mixture which consists of 0.8% potassium oxalate and 1.2% ammonium oxalate. 0.1 ml of this solution is required for each 1 ml of blood.

Tests on stools It is quite satisfactory to send samples of stools by road or rail if they are to be examined for cysts of protozoa or ova of helminths. Examination for free forms (trophozoites) of *Entamoeba histolytica* (e.g. in mucus passed per rectum or in rectal scrapings) should be carried out without delay and specimens should not be sent to a distant laboratory.

Threadworm ova Stool examination is useless for the demonstration of threadworm ova. A swabbing of the perianal region should be carried out in the morning before the patient has

had a bath and for this purpose a throat swab dipped in a small quantity of normal saline will be found useful. The swab is shaken in the saline after it has been applied to the anal region several times and the saline (containing the ova) is sent to the laboratory.

Worms or segments of worms These can be sent to the laboratory in normal saline solution if they can be examined within about 24 hours. If a longer time must elapse before examination the specimens should be sent in formal saline.

Biopsy specimens These should be sent to the laboratory in formal saline and it is immaterial how long the journey takes.

Skin tests These may be of help in diagnosis so long as it is remembered that a positive intradermal test is evidence of past as well as of present infection. In each case 0.1 ml of antigen is injected intradermally using a Mantoux syringe and the smallest hypodermic needle. The reactions are as follows:

Filariasis Schistosomiasis Hydatid disease	}	The original weal doubles in size after 20–30 minutes. There may be pseudopodia in addition.
Histoplasmosis Blastomycosis Coccidioidomycosis	}	An area of erythema and induration at least 5 mm in diameter should appear by 48 hours.
Brucellosis		At 48 hours there is a red indurated plaque 2–6 mm in diameter which may persist for several days.
Espundia		A papule develops and reaches its maximum size by about 48 hours. It remains for a few days (Montenegro's test).
Lymphopathia venereum (climacicubus)		Reaction at 24–48 hours and reaches its maximum in 3–4 days. It takes the form of a papule, vesicle or pustule with a diameter larger than 6 mm (Frei test).

DISEASE	MATERIAL	EXAMINATION FOR
Actinomycosis	1 Discharge from a fistula 2 Aspirated material 3 Biopsy 4 Sputum (if thoracic disease)	<i>Actinomyces bovis</i> do do Granules
Amoebiasis	1 Stool 2 Mucus Rectal scrapings	Cysts of <i>E histolytica</i> Free forms (trophozoites) of <i>E histolytica</i>
Ancylostomiasis	3 Pus from liver abscess Stool	do Hookworm ova
Ascariasis	Stool	Roundworm ova
Bacillary dysentery	Stool	Pathogenic bacteria
Balantidiasis	Stool	<i>Balantidium coli</i>
Bartonellosis		
a Noneruptive stage (Oroya fever)	Blood films	<i>Bartonella bacilliformis</i>
b Eruptive stage (Verruga Peruana)	Venous blood (for culture) Biopsy	do do
Bejel — see Endemic syphilis		
Bilharziasis — see Schistosomiasis		
Blastomycosis		
a North American blastomycosis	Blastomycin (antigen for skin test) Aspirated material Scrapings from skin lesions Biopsy Sputum Urine Cerebrospinal fluid Serum	Reaction to intra dermal test <i>Blastomyces dermatitidis</i> do do do do Complement fixation test
b South American blastomycosis	Antigen for skin test Aspirated material Scrapings from lesions Biopsy Sputum Serum	Reaction to intra dermal test <i>Blastomyces brasiliensis</i> do do do Complement fixation test

DISEASE	MATERIAL	EXAMINATION FOR
Brill's disease — see Typhus Group		
Brucellosis	Brucellin (antigen for skin test) Blood (for culture) Blood (for guinea pig inoculation) Serum	Reaction to intradermal test <i>Brucella</i> organisms do Agglutination reaction
Chagas's disease — see Trypanosomiasis		
Cholera	Stool	<i>Vibrio cholerae</i>
Chromoblastomycosis	Biopsy	Fungus
Cysticercosis	Cyst removed from tissues	Scolex
Clonorchiasis	Stool	Ova of <i>Clonorchis sinensis</i>
Coccidioidomycosis	Coccidioidin (antigen for skin test) Discharges or scrapings from skin lesions Biopsy Sputum Serum	Reaction to intradermal test <i>Coccidioides immitis</i> do do Complement fixation test
Dracontiasis	Fluid from vesicle	Embryos of <i>Dracunculus medinensis</i>
Dysentery — see Bacillary or Amoebic dysentery		
Endemic syphilis	Fluid from ulcer or moist lesion Biopsy Serum Serum	Treponemes by dark ground illumination Histological changes and presence of treponemes Serological reactions such as the VDRL test the Kahn test and the Wassermann reaction Treponemal immobilization (TPI)
Enteric fever — see Typhoid		
Enteritis	Stool	Pathogenic bacteria
Enterobiasis	Peri anal swabbing	Ova of threadworm
Espundia — see Leishmaniasis		

DISEASE	MATERIAL	EXAMINATION FOR
Filariasis		
a Tests for the whole filarial group	Antigen for skin test	Reaction to intradermal test
	Serum	Complement fixation test
b Bancroftian filariasis	Night blood (blood films or 5 ml in oxalate bottle) Day or night blood is suitable in the Pacific	Microfilariae (embryos) of <i>Wuchereria bancrofti</i>
c Loiasis	Day blood	Microfilariae of <i>Loa loa</i>
d Onchocerciasis	Skin shavings	Microfilariae of <i>Onchocerca volvulus</i>
e Streptocerciasis	do	Microfilariae of <i>Dipetalonema streptocerca</i>
Gastro enteritis — see Enteritis		
Giardiasis	Stool	Cysts of <i>Giardia intestinalis</i>
Glandular fever	Serum	Paul Bunnell test
Granuloma venereum (ulcerating granuloma of the pudenda)	Biopsy	Histology Donovan bodies
Guinea worm — see <i>Dracontiasis</i>		
Histoplasmosis	Histoplasmin (antigen for skin test)	Reaction to intradermal test
	Blood films	<i>Histoplasma capsulatum</i>
	Sternal marrow	do
	Sputum (if pulmonary involvement)	do on culture
	Morning sputum collected in a sterile bottle after the patient's mouth has been thoroughly cleaned	
	Gastric washings	<i>H. capsulatum</i> on culture
	Lymph gland (removed surgically)	<i>H. capsulatum</i>
	Biopsy of skin lesion	do
	Serum	Complement fixation test

DISEASE	MATERIAL	EXAMINATION FOR
Hydatid disease	Antigen for skin test	Reaction to intradermal test (Casoni test)
	Serum	Complement fixation test
	Fluid from hydatid cyst	Scolices
Infectious mononucleosis — see Glandular fever		
Kala azar — see Leishmaniasis		
Lambliasis — see Giardiasis		
Leishmaniasis		
a Visceral leishmaniasis (kala azar)	Blood films	Leishman Donovan bodies
	5 ml venous blood in an oxalate bottle	do
	Gland juice obtained by needle puncture	do
	Sternal marrow	do
	Material obtained by spleen or liver puncture	do
b Oriental sore	Fluid aspirated from the indurated tissue at the side of the sore	do
	Biopsy of tissue at edge of sore	do
c Erythema	As for oriental sore	do
	Curettage of nasal mucosa	do
	Antigen for skin test	Reaction to intradermal test (Montenegro's skin test)
Leprosy	Skin smears	Acid fast bacilli
	Skin biopsy	{ Typical histology Acid fast bacilli
	Nerve biopsy	
	Nasal scrapings need not be carried out as it is preferable to make smears from skin lesions. The lepromin test has no place in diagnosis and should be used only for classification.	

DISEASE	MATERIAL	EXAMINATION FOR
Leptospirosis	5 ml venous blood in an oxalate bottle Venous blood	Leptospire after centrifugation Leptospire on culture or guinea pig inoculation
	The above tests must be carried out during the first 4-5 days of the illness	
	Serum	Agglutination test
	Urine collected after the 10th day of the illness and before the end of the 4th week	Leptospire
	If the urine has to be sent away for examination it should be rendered slightly alkaline as soon as it has been voided	
<i>Loiasis</i> — see <i>Filariasis</i>		
Lymphopathia venereum (climatic bubo)	Antigen for skin test	Reaction to intra dermal test (Frei test)
	Serum	Complement fixation test
Maduromycosis — see <i>Mycetoma</i>		
Malaria	Blood films	Malarial parasites
Melioidosis	Blood	<i>Pfeifferella whitmorei</i> on culture
	Serum	Agglutination test
	Urine (mid stream)	<i>Pfeifferella whitmorei</i> on culture
Mycetoma	Discharge from a fistula Aspirated material Biopsy of lesion	Fungus do do
Njovera — see <i>Endemic syphilis</i>		
Onchocerciasis — see <i>Filariasis</i>		
Onyiasis	Blood	Thrombocytopenia
Oriental sore — see <i>Leishmaniasis</i>		
Oroya fever — see <i>Bartonellosis</i>		
Paragonimiasis	Sputum	Ova of <i>Paragonimus westermani</i>
	Stool	do
Paratyphoid — see <i>Typhoid</i>		
Piedra (<i>Tinea nodosa</i>)	Affected hairs	<i>Trichosporon beigeli</i> in white nodules <i>Piedraia hortai</i> in black nodules

DISEASE	MATERIAL	EXAMINATION FOR
Pinta	Scrapings from lesions	Trepanemes by dark ground illumination
Plague	Biopsy and serum tests as for endemic syphilis	
	Aspiration of bubo	<i>Pasteurella pestis</i>
Psittacosis	Blood	do on culture or guinea pig inoculation
	Sputum (in pneumonic plague)	<i>P. pestis</i>
	Blood	Psittacosis virus on mouse inoculation or culture
	Sputum	do
Q fever	Serum	Complement fixation test
	Sputum	<i>Rickettsia burneti</i> on culture or animal inoculation
Rot bite fever	Serum	Complement fixation test
a Sodoku	Blood during febrile phase	<i>Spirillum minus</i> on guinea pig inoculation
b Haverhill fever	Aspiration of lymph gland	
	Blood during febrile phase	<i>Streptobacillus moniliformis</i> on mouse inoculation
Relapsing fever	Blood films during febrile phase	The specific spirochaetes
Rhinoscleroma	Biopsy of granuloma	Histology Presence of <i>Klebsiella rhinoscleromatis</i>
	Serum	Complement fixation test
Rhinosporidiosis	do	Agglutination reaction
	Biopsy	Typical sporangia filled with spores (<i>Rhinosporidium seeberi</i>)
Rickettsialpox — see Typhus Group		
Schistosomiasis	Stool	Schistosome ova
	Urine (end specimen)	do
	Rectal snips	do
	Serum	Complement fixation test
	Antigen for skin test	Intradermal reaction

DISEASE	MATERIAL	EXAMINATION FOR
Scrub Typhus — see Typhus Group		
Smallpox	Smears made from the juice obtained from scraping a papule or the base of a vesicle Aspiration of a vesicle or pustule Scabs All the above materials	Elementary (Paschen) bodies Culture of virus (hen's egg) do Complement fixation test Flacculation test
Strongyloidiasis	Stool	Larvae of <i>Strongyloides stercoralis</i>
Taeniasis	Segments Stool	Identification (<i>T. solium</i> , <i>T. saginata</i> or <i>Diphyllobothrium latum</i>) Ova of <i>Hymenolepis nana</i> (dwarf tape worm)
Tapeworm — see Taeniasis		
Threadworms — see Enterobiasis		
Tick Typhus — see Typhus Group		
Tinea infections	Skin scrapings	Fungus
Toxoplasmosis	Serum	Complement fixation test and dye test (Sabin test)
	Both the C F T and the dye test should be positive before diagnosing active infection	
Trichomycosis axillaris	Affected hairs	<i>Corynebacterium tenuis</i>
Trypanosomiasis a African	Blood films 5 ml oxalated blood Lymph gland juice obtained by needle puncture 2 ml cerebrospinal fluid	Trypanosomes (<i>T. gambiense</i> & <i>T. rhodesiense</i>) do do Trypanosomes in creased total protein Increased globulin Increased leucocytes Decreased chloride and glucose

DISEASE	MATERIAL	EXAMINATION FOR
b South American (Chagas's disease)	5 ml oxalated blood	<i>Trypanosoma cruzi</i>
	Blood	do after guinea pig inoculation
	Blood	Trypanosomes in reduviid bugs after feeding on the blood (xenodiagnosis)
	Serum	Complement fixation test (Machado reaction)
Tropical Ulcer	Smears made after swabbing ulcer	Spirochaetes and fusiform bacilli
Tularaemia	Blood	Inoculation of mouse or guinea pig
	Serum	Agglutination reaction
Typhoid and Paratyphoid	Stool (during first 2 weeks)	Pathogenic bacteria
	Blood	Culture
	Serum	Widal reaction
	Urine (after 14th day)	Culture
Typhus Group		
a Epidemic typhus	Serum	Weil Felix reaction
		Rickettsial agglutination reaction
		Complement fixation test
b Brill's disease	As for epidemic typhus	
c Murine typhus	do	
d Scrub typhus	do	
e Tick typhus	do	
f Rickettsialpox	do	
	Note: The Weil Felix reaction is negative in rickettsialpox	
Ulcerating granuloma of the pudenda — see Granuloma inguinale		
Ulcus tropicum — see Tropical ulcer		
Undulant fever — see Brucellosis		
Veld sore	Exudate	Streptococci and staphylococci
		Sometimes <i>C. diphtheriae</i>
Weil's disease — see Leptospirosis		
Yaws	As for Endemic syphilis	
Yellow fever	Blood (during first 3 days)	Inoculation of mouse
	Serum	Mouse protection test
	Note: This test is negative during the illness and positive during convalescence after which it remains positive	
	It is of value in making a retrospective diagnosis	

INDEX

- Abscess liver 72 74 75 80
 - lung 62 95
 - perinephric 70
 - spleen 79
 - sub phrenic 75
- Acholic familial jaundice 77
- Achondroplasia 98
- Acromegaly 8 31 38 68 98
- Actinomycosis caecal 72 83 84
 - hepatic 76
 - pulmonary 63 70 95
- Addison's disease 33 98
- Adie's syndrome 27
- Agranulocytosis 34
- Ainhum 105
- Albers Schonberg disease 79
- Alcohol poisoning 10 25 27 76
- Alkaptonuria 28
- Alapicq 16 17
 - areata 16
- Amoebiasis caecal 72 83
 - hepatic 72 74 75 80
 - intestinal 75 81 85
- Amoeboma 81 85
- Amphoric breath sounds 64
- Amyloidosis 77 80
- Anaemia heart failure in 58
 - iron deficiency 79 103 107
 - Mediterranean 79
 - pernicious 15 79
 - sickle cell 77 79 83 98 112
 - von Jaksch's 79
- Aneurysm aortic 27 48 52 61
- Ankylosing spondylitis 41 68
- Antimony poisoning 76
- Anus fistula of 94
 - fissure of 94
- Aortic valve disease 42 48 51 52 53
 - 56 57 62
- Apex beat 53
- Aphthous ulcer 31 34
- Appendicitis 75 82 83
- Arachnodactyly 98
- Argyll Robertson pupils 20 27
- Argyria 13 35
- Ariboflavinosis 24 25 29
- Arsenic poisoning 76 99
- Arthritis degenerative 8 96 97
 - gonococcal 98
 - rheumatoid 45 80 96
- Ascites 71 86
 - chylous 87
- Asthma 47 61 65
- Atebrin side effects 11 25 35 103
- Atherosclerosis 8 42 50
 - coronary 50 51 53
- Atrial septal defect 54 56 98
- Auricular fibrillation 52, 59
- Austin Flint murmur 57
- Bacterial endocarditis 79 95 104
- Behcet's syndrome 34
- Bejel - see syphilis endemic
- Bell's palsy - see facial palsy
- Beriberi 8 19 25 58 59 107 115
- Bilharziasis - see schistosomiasis
- Bitot's spots 24
- Blepharitis 18
- Botulism 25 26
- Bradycardia 50
- Breast tumour of 67
- Bronchial breathing 64
- Bronchiectasis 62 95
- Branchitis 61 62
- Branchopneumonia 62
- Brucellosis 76 79
- Bubo climatic - see lymphopathia venereum
- Bubonic plague - see plague
- Bulbar palsy progressive 30 31
- Calabar swelling 19
- Cancrum oris 34
- Carcinoma of breast 67
 - of bronchus 61 63 95
 - of caecum 83
 - of colon 82 84
 - of liver 75 78
 - of pancreas 73
 - of rectum 94
 - of stomach 74 80
 - of tonsil 37
- Carbon tetrachloride poisoning 76
- Carotenaemia 11
- Cavernous sinus thrombosis 21
- Cerebellar neoplasm 10 25 26
- Cervical spondylosis 41
- Chagas's disease 18 50 51 52 59
- Cheilosis 29

- Cheyne Stokes respiration 60
 Chiclero ulcer 28
 Chigger flea 8 105
 Chloroquine side effects 16 25
 Cholecystitis 73 74
 Cholera 10 116
 Chorea Huntington's 8
 Sydenham's 8
 Chvostek's sign 101
 Claw hand 101
 Clonorchiasis 76
 Clubbing of fingers 62 95
 of toes 62 95
 Coarctation of the aorta 42 48 52
 Condylomata acuminata 91
 syphilitica 91
 Conjunctivitis 18 21 22
 Contracture congenital 100
 Dupuytren's 100
 Coral ulcer 112
 Corrigan (collapsing) pulse –
 see water hammer pulse
 Caxa valga 8
 vara 8
 Crepitations 65
 Cretinism 31 98
 Crohn's disease 84 95
 Cushing's syndrome 49 72 81
 Cyanosis 13 29
 Cyst ovarian 71 85 87
 pancreatic 81
 Cystine disease –
 see Fanconi syndrome
 Dactylitis 96
 Dementia paralytica 10 27 32
 Dengue fever 21 99
 Dermal leishmanoid –
 see leishmaniasis
 Dermatitis herpetiformis 34
 Diabetes 11 60 106
 Diaphragmatic paralysis 72
 Dirotic pulse 52
 Dig talis poisoning 50 51
 Diphtheria 25 36 51
 D plegia congenital 8
 Disseminated sclerosis 8 10 26
 D vert culitis 81 84
 Dupuytren's contracture 100
 Dysentery amoebic 81 84
 bacillary 81 84
 Dystrophia myotonica 7
 Ectropion 18
 Elephantiasis of leg 8 112 113
 of scrotum 91
 Emetine nails 103
 Emphysema 47 48 61 64 65
 Endomyocardial fibrosis 56 58 59
 Entropion 18
 Epidemic dropsy 107
 Erythema nodosum leprosum 45 90
 93
 Espundia 34 40
 Eunuchoidism 10 15 67
 Exophthalmos 21 25
 malignant 18 21
 Extrasystoles 51 52 59
 Facial hemiatrophy 31
 palsy 18
 Fanconi syndrome 68 77
 Fascioliasis 76
 Favus 17
 Felty's syndrome 80
 Filariasis bancroftian 87 88 91 92
 113
 Fluorosis 35
 Friedreich's ataxia 8 10 50
 Functional cardiac murmurs 54 55
 56
 Galactosaemia 77
 Gall bladder distended 73
 Gangosa 35
 Gargoylism 76 80
 Gastric ulcer 73
 Gastro colic fistula 73
 Gaucher's disease 23 76 80
 Gingivitis 36
 Glandular fever 36 43 79 88
 Glaucoma 22
 Goundou 35
 Gout 28 97 106
 Graham Steell murmur 56 57
 Granuloma venereum 90
 Guinea worm 112
 Gynaecomastia 67
 Habit spasm (tic) 7 41
 Haemochromatosis 13 34 78
 Haemorrhoids external 94
 Hand Schuller Christ and sease
 21 76 80

- Heart block 50 59
 failure congestive 32 42 48 54 65
 76 79 86 92 95
 in anaemia 58
 in beriberi 58
 in Chagas's disease 59
 in endomyocardial fibrosis 58 59
 in schistosomiasis 59
 in toxoplasmosis 59
- Heat stroke 21 116
- Heberden's nodes 96 97
- Hebra nose 38
- Hemeralopia 24
- Hemihypertrophy 98
- Hepar lobatum 75
- Hepatitis infectious 76 79
 amoebic — see amoebiasis
- Hepatolenticular degeneration 78
- Hernia incisional 72
 inguinal 91
 umbilical 72
- Herpes febrilis (simplex) 30
- Hip arthritis 8
 congenital dislocation 8
- Histoplasmosis 76 80
- Hodgkin's disease 44 45 80 95
- Hookworm infection 107
- Horner's syndrome 19
- Hutchinson's teeth 23 35
 triad 23
- Hydatid disease 63 76 80
- Hydrocele 91
- Hypercalcuria 68
- Hyperkeratosis 99
- Hypertension 52 54 62
- Hypertrophic pulmonary osteoarthropathy 96 114
- Hysteria 10 20 26 41 61
- Icterus gravis neonatorum 77
- Infectious hepatitis 76 79
 mononucleosis 36 43 79 88
- Intestinal obstruction 71 72 86
- Intussusception 34 84
- Iridocyclitis 22
- Jaundice 11 74
- Jejuno colic fistula 73
- Kala azar — see leishmaniasis
- Katayama syndrome 59 61 76 79
- Kerandel's sign 18
- Keratitis 23
- Keratomalacia 24
- Keratosis solar 100
- Kidney bilharzial 81
 embryoma (Wilm's tumour) 81
 hypernephroma (Grawitz's tumour) 81
 polycystic 81
 tuberculosis of 81
- Klinefelter Albright syndrome 94
- Klippel Feil syndrome 41
- Klumpke's paralysis 101
- Koilonychia 103
- Korsakow's psychosis 58
- Kwashiorkor 7 15 76 107
- Kyphosis 68
- Larval granulomatosis 76 79
- Laryngismus stridulus 102
- Laryngitis 60
- Lathyrism 8
- Leishmaniasis cutaneous 28 110
 glandular 44
 muca cutaneous 34 40
 post kala azar dermal 13
 visceral 11 34 44 76 79 88
- Leontiasis ossium 21
- Leprosy alopecia in 17
 dactylitis in 96
 ears in 28
 erythema nodosum in 45 90 93
 eyebrows in 15
 eyes in 23
 foot drop in 8
 hands in 98 101 104
 larynx in 10
 leucodermia in 13
 nose in 34 35 38
 oedema in 19 108
 testis in 67
 ulceration in 112
- Leptospirosis 21 76 114
- Leucodermia 13
- Leukaemia 34 36 43 45 77 80
- Leukoplakia 32
- Lichen planus 33 103
- Linea nigra 72
- Lineae albicantes 72
 atrophiae 72
- Liver amoebic abscess 72 73 74 76
 80
 carcinoma 75 78
 cirrhosis 75 79 86 99 107
 enlargement 76

- Liver fatty infiltration 76 80
 in protein deficiency 80
 in sarcoidosis 78 80
 in schistosomiasis 76 79
 in tertiary syphilis 75
 Löffler's syndrome 61 76 79
 Loiasis 19
 Lordosis 69
 Lung abscess 62
 Lupus erythematosus 17 28 30 40
 45 77 80
 vulgaris 28 34
 Lymphadenoma 44 45 80 95
 Lymphadenopathy 43
 Lymphoedema 113
 Lymphoedema praecox 113
 Lymphopathia venereum
 (lymphogranuloma inguinale
 climatic bubo) 84 88 90 94
 Lymphostatic verrucosis 107

 Malabsorption syndrome 68 71 72
 73
 Malaria 52 76 79 83 116
 Mastitis chronic 67
 Meigs's syndrome 86
 Melanosis 13
 Mepacrine side effects 11 25 35 103
 Mercury poisoning 32
 M'Graine ophthalmoplegic 20 26
 Milroy's disease 113
 Miosis 27
 Miracil D 11
 Mitral valve disease 52 53 54 56 57
 59
 Mönckeberg's sclerosis 53
 Mongolism 98
 Moniliasis 33 104
 Montgomery's tubercles 67
 Morphine poisoning 27 60
 Mossy foot 107
 Muscular dystrophy 69
 Myasthenia gravis 70 26
 Mycetoma (Madura foot) 106
 Mydriasis 27
 Myeloscclerosis 79
 Myotonia atrophica 7 50
 Myxoedema 10 15 17

 Nails Atebrin staining 103
 emetine 103
 Nails in acute trichinosis 104
 in anaemia 103
 in eczema 104
 in leprosy 104
 in lichen planus 103
 in melaniasis 104
 in psoriasis 104
 in Raynaud's disease 104
 in ringworm infection 104
 in sclerodactyly 104
 in scurvy 104
 in secondary syphilis 104
 in subacute bacterial endocarditis
 104
 Neuritis peripheral 58 115
 Neurosis 7 32 98
 Niemann-Pick's disease 76 80
 Noma (cancrum oris) 34
 Nystagmus 26

 Ochronosis 28
 Oedema of eyelids 18 19
 of feet 107
 Onchocerciasis iridocyclitis in 22
 keratitis in 23
 scrotum in 93
 skin in 70 114
 Onyafai 32
 Oriental sore — see leishmaniasis
 Osteitis deformans (Paget's disease)
 25 68
 fibrosa cystica 68
 Osteomalacia 8 68
 Osteopetrosis 79
 Osteoporosis 68
 Otorrhoea 28
 Ovarian cyst 71 85 87
 tumour 71 86
 Oxycephaly 21

 Paget's disease — see osteitis
 deformans
 Pancreas carcinoma of 73
 cyst of 81
 Paragonimiasis 62 63 95
 Paralysis agitans 8
 Paralytic leus 71
 Paronychia 104
 Patent ductus arteriosus 53 54 56
 Pellagra 30 42
 Pemphigus vulgaris 34
 Pericardial friction 57
 Pericardial constriction 79 86

- Peripheral neuritis* 58 115
Peritonitis 72 73 86
Perthes's disease 8
Pertussis 32 61
Phrynoderma 24
Piedra 16
Pinguecula 23
Pinta 13 99
Plague 21 36 44 62 88
Pleural friction 65
Pneumonia lobar 61 62 65
 viral 61
Polycythaemia rubra vera 14 29 79
Polypoid intestinal 84 95
Porphyria 35
Post kala azar dermal leishmaniasis —
 see leishmaniasis
Pregnancy 8 13 67 71 72 85 108
Pseudobulbar palsy 10 31
Psoriasis 104
Pterygium 23
Ptosis 19 20
Pulmonary oedema 60 62
 stenosis 53 56
Pulsus alternans 52
 bigeminus 51
 bisferiens 52
 paradoxus 52
Purpura haemorrhagica 79
Pyloric obstruction 72 74

Q fever 61

Rales 65
Raynaud's disease 98 104
Regional enteritis 84 95
Relapsing fever 22 76 79
Resochin side effects 16 25
Rhinophyma 38
Rhinoscleroma 38
Rhinospandiosis 38
Rhonchi 65
Rickets 47 68 102
 renal 68
Riedel's lobe 73
Ringworm — *see tinea*
Risus sardonius 14
Romana's sign 18
Rosacea 38 49
Rubella 43

Salpingitis 84
Sandfly fever 21
Sarcoidosis 18 44 78 88
Schistosomiasis 76 79
 skin lesions in 91
 pulmonary hypertension in 59
Sclerodactyly 99 104
Scleroderma 99
Scoliosis 68
Scrotum 91 92 93
Scurvy 25 36 96 104
Scybalum 81 85
Septicaemia 79
Sibili 65
Sickle cell anaemia 77 79 83 98 112
Siderosis 77
Simmonds's disease 15
Sinus arrhythmia 51
Skodaic resonance 64
Snake bite 25
Soft sore 88 90
Spastic colon 85
Spermatic cord 92
Spider naevi 49 99
Spina bifida cystica 69
 occulta 69
Spleen enlargement 79 80
Sporotrichosis 112
Sprengel's shoulder 69
Sprue syndrome 11 30 31 68 71 72
 73 86 95 102
Sputum 66
Steatorrhoea — *see sprue syndrome*
Still's disease 80
Stokes Adams attacks 50
Strabismus 25
Stridor 60
Strychnine poisoning 14
Succussion splash 65
Syphilis congenital 23 35 38 40 79
 91 96
 endemic 35 91 99
 primary 31 88 89 91
 secondary 17 25 31 35 37 44 89
 91
 tertiary 25 31 75
Syringobulbia 10 26 31
Syringomyelia 26 96

Tabes 8 20 27 106
Tapir nose 40

- Telangiectases 49
- Testis atrophy of 93 94
 - enlargement of 93
 - inflammation of 93
- Tetanus 14 41
- Tetany 14 73 101 102
- Thyrotoxicosis 21 32 42 46 48 52 54
- Thrombophlebitis 108
- Thrombosis of portal vein 79 86
 - of splenic vein 79
- Thrush 33
- Thyroid gland enlargement 46
- Tietze's disease 48
- Tinea pedis 105
 - tan urans 17
 - unguium 104
- Tonsillitis 36
- Torticollis 41
- Toxoplasmosis 43 59 76 79
- Trachoma 18 21 22
- Trichiniasis 18 104
- Trichomycosis axillaris 16
- Trichterbrust 47
- Tricuspid valve disease 42 78 86
- Triple rhythm 54
- Trismus 14
- Tropical eosinophilia 25 61 76 79
- Trousseau's sign 102
- Trypanosomiasis African 8 18 25 27 32 44 76 79 115
- Tuberculosis cold abscess in 48 68 69 92
 - dactylitis in 96
 - glandular 44 87
 - hip joint in 8
 - intestinal 72 83 95
 - laryngeal 10
 - miliary 76
 - pulmonary 47 61 62 65 79 83 95
 - spinal 69
- Turner's syndrome 41 94
- Typhilitis amoebic 72 83
- Typhoid fever 15 52 62 73 79 81
- Typhoid enteritis 48
- Typhus fever epidemic 21 62 92
 - rub 21 43 44
 - tick borne 88 90
- Ulcer aphthous 31
 - coral 112
 - in dracontiasis
 - in late yaws 111
 - in leprosy 112
 - in sickle cell anaemia 112
 - lingual 31
 - peptic 73 86
 - trophic 106
 - tropical 110
 - varicose 112
- Ulcerating granuloma of the pudenda 90
- Ulcerative colitis 22 81 82 95
- Ulcus molle 89 90
 - molle serpigiosum 90
- Uterus fibromyoma of 71 85
 - in pregnancy 71 85
- Varicocele 92
- Varicose ulcer 112
- Veld sore 110
- Vena caval obstruction 19 29 42 48 74
- Veno occlusive disease 78
- Ventricular septal defect 53 54
- Vestibular disturbance 26
- Vincent's angina 36
- Vitiligo 13
- Van Gierke's disease 77
- Van Jaksch's anaemia 79
- Water hammer pulse 51
- Weil's disease - see leptospirosis
- Wernicke's encephalopathy 25 58
- Whipple's disease 45
- Whispering pectoriloquy 65
- Wilson's disease 78
- Winterbottom's sign 18 44
- Xanthelasma palpebrarum 11
- Xanthomatosis 11
- Xerophthalmia 24
- Yaws crab 8 111
 - dactylitis in 96
 - early 44 99 100 111
 - late 13 35 99 111
- Yellow fever 21 76

